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MAORI ATTITUDES TO SICKNESS.[1]

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Maori Concepts of Sickness.

THE old-time Maori believed the principal causes of illness or death to arise from violation of tapu, from the intervention of Atua or dæmons, or not infrequently by machinations of the Tohunga Makutu.[2] Causes of death were grouped under the following three main attributions: (i) Mate atua or Mate Maori, death due to intervention of atua or "dæmons"; (ii) Mate Taua, death in battle; (iii) Mate Aitu, death due to natural causes or "death by the wall of the house".

Conversion to Christianity and the impact of European colonization have by no means eliminated either the attitudes or the practices deriving from these beliefs. There is at the present time considerable evidence of a revival in Maori recourse to the Tohunga, or one claiming skills in ancient lore, and to many practices deriving from the "classical" age of Polynesian culture in New Zealand—that is to say, the period of Cook's voyages.

Maori medicine, both in the "classical" period and today, contains within its corpus two distinct elements. The first is the system of magical practices designed to discover or counteract the effects of a breach of tapu, or a malignant mana which supposedly lay at the root of sickness. The second is a miscellany of herbal medicines and physical methods of treatment, some accurately based on the pharmaceutical properties of native plants, others deriving from folk lore and custom. The efficacy of some of these preparations and methods sufficiently impressed the early colonists to lead to their adoption by survey parties and rural settlers—a fact which is also a commentary on European medicine of the 1840's.

A passing mention may here be made of a number of the traditional vegetable preparations and physical methods of treatment; a very full list of these is to be found scattered through the writings of Elsdon Best, Gudgeon and other writers.

For scabies, there were varied vegetable remedies reinforced by ritual and generally ineffectual in practice. The use of hot springs and thermal mud was more effective.

For diarrhœa, Manuka, Toitoi and Koromiko were used, the last-mentioned being very useful for mild dysenteries.

Constipation was usually due in former times to eating fern root. Various methods were devised to remove hard stools mechanically by means of shaped sticks, *et cetera*.

[1] Based on a paper read at a meeting of the Victorian Council for Mental Hygiene on October 17, 1955.

[2] Tohunga Makutu was a "skilled man" trafficker in evil or malignant mana.

Hæmorrhoids were treated by Totara smoke, over which the victim sat, the smoke being directed up a narrow vent.

In the repair of fractures, bark of hoheira or lace-bark was used in a similar manner to Gooch splinting. This is very efficacious and readily procurable, there being many species of closely allied trees which give a useful bark for the purpose.

In the treatment of convulsions from Karaka poisoning, burial of the afflicted person up to the neck in sand or loose soil was a popular method of minimizing injury. Recovery followed if the victim survived for twenty-four hours.

In resuscitation of the partially drowned, both artificial respiration and elimination of inhaled water were attained by suspending the affected person by the legs round the neck of a large warrior who, grasping the wrists, moved the person up and down. The resultant movement resembles the rocking method of resuscitation. The head was suspended over Totara smoke if such a fire could be made.

Among child-birth practices there were a number of traditional manœuvres to assist difficult labour and to aid the bearing-down movements. There is, however, no evidence of the old Marquesan custom of sucking the breasts and of sexual intercourse to combat post-partum hæmorrhage.

Reinforcement of drugs by charms was employed. Drugs were sometimes carried in containers bound round with tokens of high potential mana. An example of this is a koromiko container bound with human hair.

The Maori attitude to the sick person was dependent upon the strongly held beliefs in the causation of illness by varied powerful forces not wholly controllable by the learned and skilful Tohunga, and even less so by the ordinary person.

The apparent Maori neglect of the sick, which was very pronounced in former times, is usually a logical consequence of their ideas of the causation of sickness—not of callous indifference, as some Europeans assert. Sickness was normally thought to arise most commonly from a breach of tapu. In consequence, logically enough, the sick person would be a source of danger. The death of the sick person would render the locus even more highly tapu. When the death occurred in a dwelling, for example, the dwelling would have to be destroyed or abandoned. Hence arose the custom of placing the sick in tents or outhouses, structures of little value. The contaminated ground could be dealt with later or just avoided. According to the Tohunga and to common beliefs, an unexpected death was a stroke of misfortune or an outcome of revenge-directed malignant mana, or else was due to the action of the deceased himself by breach of tapu.

Mana.

Some parts of the body were regarded as being more highly tapu than others. Certain parts of the body had a beneficial mana, others a dangerous mana. The Maori, in common with many other Polynesian groups such as the Marquesans, believed in the destructive or dangerous mana inherent in the female organs of generation. (Compare sayings of Jesus, "I have come to destroy the works of the female"; this appears in "The Gospel According to the Egyptians", quoted by Clement of Alexandria.) All female generative secretions were regarded as carrying noxious or harmful mana. Until very recent times, one of the Maori objections to entering European maternity hospitals was related to their consequent inability to deal with the afterbirth in the traditional manner by burial in a secret place. Incineration or burning of the placenta was believed harmful to the child. Menstrual fluids and women's urine were regarded logically as having the malignant properties of the organs through which they passed.

The Maori male organ, however, represented life. It played a prominent part in many magical practices. Logically enough, urine which passed through the male organ was believed to acquire valued properties. Therefore, the village latrine was a site of very great—manifest and perceptible—mana. Thus ceremonies for protection against disease and danger in time of trouble, or at the start of a journey, *et cetera*, were often carried out at the village latrine. Especially binding oaths were sworn with the penis held in the left hand.[1]

Maori Values in Community Life.

Here it may be as well to consider a few characteristics of the old-time Maori which are necessary to the understanding of the present-day persistence, or active revival, of Tohungaism and recourse to varied practices, some magical, some traditional and some herbal, to which I shall later refer.

The focal point of the Maori village was and is the marae, the open space in front of the meeting house, the supports of which were covered with ancestral figures—some of Maori forebears, others of mythological ancestors of the tribal group. The Maori attaches great importance to kinship and the family. The group and community centering around a common Marae are strongly linked and mean much. They are important not only to work, but also to much wider social activities. The prospect of tribal gatherings, and other social occasions, act as a stimulus to economic effort. No one who has had the privilege of attending a large Maori gathering, such as Koroki's Coronation anniversary at Turangawaewae, could maintain the popular though erroneous European belief that the Maori is and always has been lazy. Maori gatherings demand the accumulation, preparation and serving of immense quantities of food, and considerable ingenuity is displayed in this large-scale communal enterprise. For example, one of the problems at Turangawaewae was cooking potatoes in quantities of up to one ton and a quarter at a time. It was solved by an ingenious system of steam-heated containers made from disused water tanks. Preparations are made well in advance for the necessary supplies of pigs. These are fattened on widely scattered farms and collected at the appropriate time by a Maori who, in one important group, is scarcely literate, yet will collect all his fattened animals without the aid of a single note. A capacity to commit lengthy lists of ancestral names to memory is doubtless related to such aptitudes and is not dependent on "literacy".

The family and tribal group was of supreme importance to the old-time Maori. An almost equal value attached to the ancestral lands.

Sexual Mores and Customs.

In the field of sexual and reproductive mores, the Maori followed a common Polynesian pattern. There was little or no hindrance to pre-marital sex relations between adolescents. Though the Maori did not actually encourage infantile sexuality or set a value on erotic training of his women on the elaborate pattern of the Marquesans, he was, like the Marquesan, fully aware of infantile sexuality centuries before Freud. Pre-marital relations were accepted as one of the pathways to marriage, and were not normally regarded as a violation of their easy sexual or marital code. Children were welcomed and freely adopted. Therefore it is not surprising to find, at the present time, little conflict over illicit pregnancy. Until very recently, Maori recourse to abortion or contraception was comparatively rare. Traditional Maori aversion to contraception is on totally different grounds from those of the Roman Church. The Maori attitude is very easy with regard to child adoption, whether temporarily or more or less permanently. The exchange of children for varying periods was and is by no means uncommon, although it never attained the proportions that it did in the Marquesas. After marriage, a reasonable measure of fidelity is expected. Women past child-bearing are often tolerant of periodic "lapses" by their husband.

With the vigorous persistence of these patterns, there need be no surprise at the present remarkable increase in

[1] It is interesting to note that in Spain, as late as the early eighteenth century, the emanations from excreta were regarded as having an essential influence on the fermentation of yeast, and a law was passed compelling all bakehouses to incorporate a privy.

the Maori population. It is more than sufficient to overcome the high Maori infantile mortality rate. I would suggest that the relatively high Maori infant mortality is probably due to the persistence of certain Polynesian attitudes to disease and infection where child-rearing practices are concerned.

After an initial period of intense spoiling and petting, the Maori child is allowed very great freedom as judged by European standards. It tends to run very much with its own age group, a child sometimes only two or three years older being delegated the responsibility of keeping the younger siblings out of more obvious danger. When the mother is working, the grandmother or an older female relative eagerly takes over a measure of responsibility for the child. The grandmother may often exercise influence and control over such matters as the naming of her grandchildren and the exhortation of daughters-in-law who show signs of slackening procreativity. In bygone days Granny's thigh bone was a potent fertility charm if she herself had been fertile beyond the norm. Grandmothers play quite a large part in Maori mythology and in the practice of magic. They have also played conspicuous parts in tribal counsels and leadership in past and in recent times.

The Tohunga and Magical Practices.

It is now perhaps as well to return to the function of magical practices in old-time Maori society and their persistence today. Magic[1] was freely employed in all fields where danger might affect the human group. The traditional herbal and practical remedies being somewhat limited in their scope, the Maori faced the problem of illness by interpreting it in such terms as breach of tapu or malevolent sorcery. Belief in curative and black magic was, and still is, in some degree widespread. The most unfortunate aspect of the present-day recourse to the Tohunga or "skilled one" is the resultant unwillingness to go to hospital or to seek European medical aid until the patient is far advanced in his disorder. Too often the patient is reluctantly sent to hospital or seeks the European doctor only when *in extremis*. Not infrequently he may die shortly afterwards, to the great satisfaction of the Maori Tohunga, who is not averse to pointing out the evil consequences which followed consultation of the European doctor. The Tohunga goes scot-free and the Pakeha physician gets the blame. What is more, there is another ready-made source of endless gossip at the *tangi*, or funeral feast.

Some indication of the methods of the present-day Tohunga may be of interest (Blake-Palmer, 1954).

> Tohungaism of to-day is seemingly eclectic and essentially debased. When firmly established in a district it often hinders the more suggestible Maori from taking advantage of the freely available services of the New Zealand hospital system. To some extent it serves to perpetuate vague fears and hostility to present day European medicine: in much the same way certain ill-informed groups in the European community prefer to foster old prejudices, fears and hostility towards modern methods when practised in State institutions for the treatment of the mentally ill.
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> Perhaps it may be well to extend this lengthy introduction by citing a few instances of the commoner "medical" practices of the present day tohunga. These methods are not in any way similar to the methods of a competent European psychiatrist, despite the assertion to the contrary by a Maori Welfare Officer in the course of a Court hearing.
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> Prominent among the more favoured methods are immersion of the sick at dawn and sundown in the wai tapu which may vary from the usual running stream to a more stagnant cattle pond. Children supposedly possessed of a Maori devil—and the reputed symptoms attributed to this complaint vary from extreme restlessness to the faulty or absent speech of the congenital mental retardate—are not infrequently chastised or locked up, apparently on the same principle that led Xerxes to order the lashing of the Hellespont after the destruction of his bridge of boats by a storm. Obviously such practices in effect may amount to—or even result in Court charges of—maltreatment or neglect. So may the comparative indifference with which many Maoris still treat the sick, especially those whose illness is accompanied by signs of mental disorder. The Maori is not neglectful of malice; he is merely avoiding possible contamination by forces which he believes responsible for the sick persons' affliction. It is dangerous to meddle in such matters. Experience proves this to the Maori's satisfaction—was not the sickness catching, or did not some misfortune later befall? Such attitudes of course are not unknown in the Pakeha community especially among the lesser and more peculiar religious sects and crank societies.
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> Some tohunga methods of treatment are frankly dangerous, especially that custom of incising the gums of toothless or teething infants with a sharp unsterilised pipi shell. Death has occurred from such "operations". All will agree such acts should not be tolerated even by the most liberal apologists for the growing pains of a Polynesian community under the impact of Western industrialism.
>
> Herbal remedies are numerous and are in themselves usually innocuous. However, they may delay the application of more efficacious European methods, and may thus lead to the spread of preventable contagion. In this glass-house, however, the European community cannot afford to cast stones, for "herbal healers" flourish. Again commercial broadcasting stations draw attention to nostrums which some Maoris may use even with a sense of superior virtue, at having thus risen above the old-time ways of their family. Indeed one man proudly boasted as evidence of his enlightenment that he has treated his child with a proprietary cough mixture (containing a high percentage of alcohol) whereas his wife had recourse to daily dippings in the wai tapu. Neither treatment was of the slightest value to the congenital disorder from which the child suffered unless the cleansing value of the wai tapu tipped the scale in favour of the older ways.
>
> An exception to this might be made in the case of a few zealous and painstaking searchers after breach of tapu (and other sins). In the course of several days "anamnesis" a valuable mental catharsis not infrequently ensues. Such practitioners are—in my experience—rare, especially in the Northern area from which the majority of my examples are drawn. . . . Nevertheless the time spent by the Tohunga is undoubtedly of solace, and more personal than most European medical practitioners can accord.

Mental Disorders in Maori Communities.

I feel sure that members of the Victorian Council for Mental Hygiene may wish to hear of the present incidence of mental disorder in the Maori communities in New Zealand. My earlier observations have, in large part, served as an introduction to this particular aspect of Maori health and Maori medicine. It would seem that the old-time Maori was not very attentive to the mentally deranged, who was frequently feared, commonly shunned and sometimes eliminated. Today the admission rate for Maoris to mental hospitals is significantly lower than that of Europeans, definitely lower than can be accounted for by reluctance to seek aid or tolerance of the high-grade defective in rural communities.

If the European rate is taken as the mean, in 1951 (the last year for which complete census figures are to hand) the Maoris might have been expected to occupy 376 mental hospital beds. In actual fact the number was 240. In 1953 the incidence of Maori admissions was 20·61 per 10,000, as compared with 47·73 per 10,000 of the European or Pakeha population. The expected number of Maori patients should have been 31·27 per 10,000. In considering these figures, one must also bear in mind that the Maori population in New Zealand is relatively a much younger population than the European or Pakeha one. For example, in the sixty to sixty-five years' age group, the Maori percentage of the population is only 2·2, whereas in the thirty to thirty-five years' age group it is 4·8 and in the twenty to twenty-five years' age group it is 7·7. On the other hand, the admission rate to Borstal institutions and prisons is relatively high, and many of these lads have a borderline intelligence quotient or less.

With regard to the type of mental disorder, there are several discernible differences between the Pakeha or European figures and those of the Maori.

[1] The term "magic" is not used in its restricted sense, but to cover the field of practice designed to counteract supposed evil influences or emanations.

1. In the sixty-five to eighty years' age groups, the Maori admission rate is lower than the estimated admission rate based on the percentage of Maoris in the group.

2. Grossly defective Maori children tend to have a higher mortality rate than grossly defective Pakeha children.

3. A higher proportion of lower grade feeble-minded Maori youths may be admitted to mental hospital only after a sentence to a Borstal institution, when an institutional "situational episode" leads to a further mental assessment.

4. The incidence of schizophrenia does not appear to differ significantly from that of the European population, though the content and form of the disorder may show some cultural patterning.

5. States of excitement, often of brief but unusual intensity, are rather commoner in Maoris than among Pakeha[1] of United Kingdom and northern European origin.

6. In depressive illnesses, and in some hallucinatory disturbances, it is wise for the psychiatrist to consider the possibility that the Maori patient may believe himself to be in danger either from a violation of tapu or more commonly nowadays from the attentions of a Tohunga. This is particularly the case if fear is a prominent symptom. Maoris usually refer to these matters as "Maori business". The psychiatrist should always make inquiry along these lines, and he will usually find that the patient's family is also persuaded as to the origin of mental symptoms in "Maori business". With practice one gets to know the hall-marks of these fears, and that is important, because it should determine a particular line of reassurance. Discussion helps; it is worth attempting. Injection of doubt may suffice to dissipate the main sources of irrational fears.

Concerning therapeutic measures that should be adopted once the Maori has symptoms he attributes to Makutu or other "Maori business", the late Sir Peter Buck gave an illustration of his own methods. During a personal discussion after a paper at the Seventh Pacific Science Congress in 1947, he recounted the following story:

> A Maori woman on her wedding night found that she was unable to pass urine. Pain and distress were considerable and she was brought across the Hokianga River to see me. After a clinical examination I realised that though there seemed to be a very simple explanation, the woman and her husband had persuaded themselves that the distressing symptom was the work of a rival. I went into the matter carefully. I gave the necessary Maori re-assurances. I followed this with instructions that she take a warm bath after the District Nurse had given her an injection of Morphine. Matters were righted well within the hour.

Protective Value of Some Maori Attitudes.

It is of some interest to inquire into the nature of any special "mental health coping mechanisms" or factors in the Maori social group which may serve to protect their mental life. There is, I am convinced, very clear evidence of such mechanisms. There are also many deep-seated satisfactions which the Maori derives from the surviving elements of his culture, which is adapting itself to technical progress. Indeed some Europeans, including anthropologists such as Professor R. Piddington, have regarded the Maori as "the only group in New Zealand boasting a culture and individual character distinctively their own". However, not all Maoris and very few Pakeha are capable of perceiving the stimulus of the "live" Polynesian thought, customs and practices which make up "Maoritanga", for which term there is no exact English equivalent. Those who cannot readily feel any deep sympathy with the Maori way of life could, I suggest, allow greater respect for the point of view of those who do. To a Maori "the return to the marae and passing into the subdued light of the meeting house, surrounded by carved figures on the supporting posts of tribal and personal ancestors brings a sense of oneness and belonging" which very few Pakeha appear to understand and still fewer to experience.

Though many things in the Maori way of life have passed away, this central core of family and kinship relations persists. There is a strong feeling of group solidarity at family and larger tribal gatherings. A great amount of time at these gatherings is taken up in a roundabout of running commentary on social affairs, and particularly in the discussion of people—what they do, what they say, how they respond. Events are important in terms of persons.

In the younger groups, there is a greater survival of respect for age and rank than is commonly supposed, and for that matter than is the case in many Pakeha gatherings. The old Polynesian custom of maintaining the spiritual prerogatives of primogeniture in chieftains' families persists. There is a revival of interest, responsibilities and powers in the Maori tribal committees, in which due deference in selection is given to this and other aspects of Polynesian custom where chieftains' families are concerned. Though the day of the big man may be over in Maori leadership, there is a continuity of representation of the older leading families on the present-day tribal committees. The chiefly functions and prerogatives have merely been spread more widely and thus brought more into line with contemporary feeling and ideas of so-called democratic management.

Gossip and Grievances as Therapeutic Agents.

A Maori gathering may be said to have lifted gossip to the dignity of a mental health "coping mechanism", which serves two purposes. On the one hand, it serves to keep in check non-conformist behaviour by a subtle application of sanctions and the verbal exploration of possible new models of behaviour. At the same time, gossip relating to the departures from the approved norm provides a speedy opportunity to vent hostility and scarcely concealed latent aggression. The Maori is adept at making grievances, which serve quite a valuable purpose to the local community, though they may well be the despair of the Maori Affairs Department. For example, there may be strong tribal pressure to hand back land which has been taken over for development under the 1951 Act. When the Maori Affairs Department shows signs of handing the land back, the community may sometimes foil all attempts to do so by failing to provide the necessary measure of agreement. This serves two purposes. It helps to preserve the sense of integrity of the Maori community, and provides excuses for non-cooperation with the Pakeha and, what is often more important, a long-lasting topic over which verbal aggression may be liberally and even venomously spilt.

Psychiatrists unfamiliar with the Maori way of life may well be excused for wonderment that there is not a far greater neurotic breakdown rate from the widespread anxiety which so many Maoris habitually experience in face of the changing *tempo* of their economic life. A recent field survey conducted for the Carnegie Social Science Research Committee by the Victoria University College, Wellington, New Zealand, has paid some attention to this. In this preliminary report, J. A. Ritchie strongly supports the viewpoints I have just expressed, that part of the unexpected success of the Maori in escaping the consequent neurotic breakdown may well be attributable to the following: (*a*) "Coping mechanisms", such as gossip and grievance manufacture, which are usually working well and are readily and immediately available. (*b*) Alcohol, which is used to excess, the "coping mechanism" being intensified during its consumption. (*c*) The orientation of the family social structure in the Maori community towards the wider kinships, and towards coping with difficulties rather than achieving a higher economic standard. A relatively low economic status is the "social price" of mental integrity.

Withdrawal Responses.

There is another very important way in which the Maori protects his mental life and integrity, particularly in the realms of social contact in the discussion group, in the more specialized fields of academic achievement, or in the face of new departures in rural economy. I refer to the almost naïve and undisguised withdrawal in the face of any suddenly presented threat. Physical threats may well produce a contrary effect. So long as the threat can be ignored, then the situation need not be faced and the underlying worry need not be resolved. When failure seems

[1] The Maori term for Europeans and members of the so-called white races.

likely, as for example in examinations, the Maori candidate all too frequently takes the view that it is better not to aspire than to aspire and fail. This particular mechanism has also been noted by psychological investigators in the course of applying intelligence tests. It is not infrequently found that children upon whose behaviour a high score might have been predicted, often fail to come up to expectations on being tested. In this respect other well-recognized Polynesian attitudes to test situations may play a part.

The Maori is always readily capable of transferring his attention rapidly away from an immediate threat or unpleasant problem. He will also politely face a reprimand or the consequences of some delinquency, confident that he may disarm his would-be castigator by a naïve and frank (though sometimes sullenly frank) admission of guilt. The Maori community understands and approves these mechanisms. They are in no sense to be regarded as a withdrawal from reality. They are more properly an expression of preference for another and pleasanter reality. Meanwhile the problem remains, and alcohol is too often a popular line of defensive therapy.

Comment.

Before drawing my scattered observations to a close, I should like to return once more to the place of the Tohunga in the present-day Maori community. By consulting the Tohunga, in preference or in addition to the European or Pakeha practitioner, the Maori can enjoy the best of both worlds to his own satisfaction. Recourse to the Tohunga may be due only to a natural desire to ensure that everything possible is done. It may sometimes be in deference to family pressure, much in the same way as nominal Christians marry in church. But in many cases the Tohunga fulfils a deep-seated need, for the Polynesian belief in atua or "dæmon" origin of sickness is by no means dead. Furthermore, in the case of the more reputable and learned practitioners, the amount of time spent in painstaking and searching inquiry for the possible breach of tapu, often over a period of several days, may well act as a health-restoring mental catharsis. European hospital treatment must mean some separation from family, and offers no comforts comparable to a credible assurance that "Maori business" is countered effectively by Maori methods. I do not think the Pakeha community can afford to take a very strong or patronizing stand in this matter. It could well make a greater effort to understand the motives which lead the Maori to consult his Tohunga, and the deep-seated, though often imperfectly understood, needs that this may serve.

Today too many self-styled Tohungas are by no means well versed in the ancient skills. Some of their "neo-Polynesian" and personal methods either are frankly dangerous in themselves, or may be harmful to both patient and community, for they rarely, very rarely, equal the prompt application of appropriate European medical methods. One can hardly expect a Director of Tuberculosis, for example, to regard the activities of some Tohungas with very much favour if their influence leads to people with open tuberculous lesions being transported up to 200 miles for the purpose of consulting one of these self-appointed and self-instructed Maori practitioners.

Another Side of Tohungaism.

Earlier I made brief reference to "black magic". This term is, strictly speaking, a misnomer, for I referred to the works of the tohunga makutu, who traffics in the manipulation of evil and malignant mana, claiming to direct it or deflect it towards the enemies or established rivals of the person who has sought his services. Recourse to such practice is by no means rare today. I have even known of one case of a sixteen-year-old secondary school girl seeking the services of such a practitioner with the intention of discomfiting her seventeen-year-old rival. Once a Maori is firmly convinced that the tohunga makutu is working on him, he may become, for the time being, extremely anxious, if not ill and indisposed. But it seldom goes any further than this. The victim usually takes prompt steps to remove himself from the danger zone, or for good measure he or she may seek the counterpoise of a more powerful operator. The continued belief in such practices may even lead to a breach of the peace. The present-day Maori, aided by alcohol, may by chance remember the effective custom of Whaka Ngu Ngu, which being freely translated reads: "Shoot the Tohunga." I know of at least two such examples, one of which was fatal—and no witnesses came forward to assist police inquiries.

Tailpiece.

Let me conclude on a more psychological note. The Maori had many proverbs. One of the sayings handed down by the old-time Tohunga is well worthy of preservation: "There is a well of dissatisfaction in the heart of man and hence vexation and anxiety."

Sources and Acknowledgements.

Apart from such general references as Elsdon Best, the source of material and observations not specifically acknowledged in the text is based upon personal observation, or has been personally communicated. I would especially acknowledge my indebtedness to Professor R. Piddington, Department of Anthropology, Auckland University College, and to Dr. Maharia Winiata, Maori Adult Education Tutor, Auckland University College, for valuable suggestions in the matter of presentation.

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A SEROLOGICAL SURVEY ON A GROUP OF NATIVES OF THE WABAG AREA OF THE WESTERN HIGHLANDS OF NEW GUINEA FOR DIPHTHERIA ANTITOXIN AND ANTIBODY TO HÆMOPHILUS PERTUSSIS.

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THE Nuffield Foundation in 1955 sponsored an Australian anthropological expedition to the Western Highlands of New Guinea, an area which carries a large native population isolated from the rest of New Guinea by the ruggedness of the terrain. The isolation is not absolute—a few prospectors, a few administration officers, and the presence of shells from the coast among the natives are evidence of some contact with the outside world.

Apart from the anthropological investigations, it was thought that a serological survey would be of interest in disclosing the infections that were present among these isolated peoples. It was already known that there was no tuberculosis, although the presence of leprosy had been reported (North and Jamieson, 1950), and that there was hardly any malaria, but there was no other definite information.

Samples of blood were flown out for the determination of the blood groups of the natives, and the sera have been made available for antibody studies.

TABLE IA.
Titrations of Diphtheria Antitoxin.

Source of Serum.	Reciprocal Titre in Bench Test (and Antitoxin Content).[1] (Units per Millilitre.)											Number of Specimens.
	<2 (<0·008)	2 (0·008)	4 (0·016)	8 (0·031)	16 (0·06)	32 (0·13)	64 (0·26)	128 (0·51)	256 (1·0)	512 (2·0)	1024 (4·1)	
New Guinea natives: adults ..	3	3	9	14	8	5	1	0	0	0	0	43
Blood donors in New South Wales, aged 18 to 59 years	35	13	11	9	7	4	0	0	0	1	0	80
Cord sera (Melbourne)[2]	30	1	9	6	4	5	5	3	1	0	1	65

	Concentration of Diphtheria Antitoxin. (Units per Millilitre.)											
	<0·01	0·01	0·02	0·04	0·1	0·2	0·5	1·0	2·0	5·0	10·0	
Cord sera (London)[3]	1029	95	105	151	166	201	122	86	35	9	1	2000

[1] Calculated from bench titres obtained in hæmagglutination test.
[2] Data of Fisher (1952).
[3] Data of Glenny *et alii* (1955).

It is the purpose of this paper to report the result of investigations of (i) the diphtheria antitoxin content of the sera, and (ii) the pertussis antihæmagglutinin content of the sera, as indications of the prevalence of infections with *Corynebacterium diphtheriæ* and *Hæmophilus pertussis* in this region. For comparison, a number of specimens of serum collected in New South Wales were also tested.

The results of other surveys carried out on the sera collected by the expedition will be reported elsewhere.

Materials and Methods.

Sera.

The sera of the New Guinea natives, and specimens of serum of blood donors in New South Wales, were supplied by Dr. R. J. Walsh and Dr. H. K. Ward, of the Blood Transfusion Service of the New South Wales Division of the Australian Red Cross Society. The sera taken in New Guinea were those of adult males whose age could not be determined with any accuracy. From New South Wales 20 specimens of serum were tested in each of the following age groups: eighteen to twenty-nine years, thirty to thirty-nine years, forty to forty-nine years and fifty to fifty-nine years; the contributions by the two sexes were similar.

TABLE IB.
Titrations of Diphtheria Antitoxin: Sera Giving Reciprocal Bench Titre of 2 or More. (Derived from Table IA.)

Source of Serum.	Proportion of Total Number of Sera in Group.	Geometrical Mean.	
		Of Reciprocal Bench Titre.	Of Diphtheria Antitoxin Concentration. (Unit per Millilitre.)
New Guinea natives ..	40/43	8·9	0·036
New South Wales blood donors	45/80	6·3	0·025
Cord sera (Melbourne) ..	35/65	18·4	0·074

Forty-three specimens from New Guinea were tested for diphtheria antitoxin, and a different series of 50 specimens was tested for pertussis antibody. In the case of the sera from New South Wales, the same series was used for the two tests.

Titration of Diphtheria Antitoxin.

The method of Fisher (1952) was used for titration of diphtheria antitoxin. It consists of hæmagglutination tests on falling dilutions of the serum to be tested against sheep erythrocytes to which tannic acid and then diphtheria toxin have been attached. The essential feature of this technique—that is, that the hæmagglutinating titre of human serum is a measure of its diphtheria antitoxin content—has been corroborated by findings of other workers (Landy, Trapani, Formal and Klugler, 1955), and is further supported by rabbit intradermal toxin neutralization tests made on pools prepared from the New Guinea sera, by means of the technique previously described (Fisher, 1952).

TABLE II.
Pooled Serum of Natives from New Guinea: Correlation between In-Vitro and Rabbit Intradermal Methods for Determining Concentration of Diphtheria Antitoxin.

Reciprocal Titre in Bench Test.	Number of Specimens of Serum in Pool.	Diphtheria Antitoxin Content. (Unit per Millilitre.)	
		Calculated from Bench Titre.	Determined in Rabbit Skin.
64 or 32	5	Between 0·13 and 0·26	Between 0·125 and 0·625
16	2	0·06	Between 0·05 and 0·25
8	9	0·03	Between 0·025 and 0·125
4	5	0·016	Between 0·005 and 0·025
2 or less	4	0·008 or less	Between 0·005 and 0·025

Tests for Antibody to H. Pertussis.

The determination of pertussis antihæmagglutinin (Warburton and Fisher, 1951) was chosen as the test for antibody to *H. pertussis*. In our hands, in tests on up to 100 serum specimens, this technique gave no less specific, and more sensitive and reproducible, results in the detection of previous contact with pertussis antigens than did titration for bacterial agglutinin (unpublished observations by G. M. McKenna and S. Fisher, 1951, 1952 and 1954).

Results.

Tests for Diphtheria Antitoxin.

The diphtheria antitoxin content in the sera from New Guinea was higher than in the series from New South Wales. In the former group, 40 out of 43 specimens (93%) contained antibody in sufficient amount to be detectable in the bench test, as against 45 out of 80 specimens (56%) in the latter. This difference is statistically highly significant ($P < 0{\cdot}01$). The mean antitoxin concentrations of the

TABLE III.
Titrations of Pertussis Antihæmagglutinin.

Source of Serum.	Reciprocal Titre.							Number of Specimens.	Specimens Giving Reciprocal Titre of 2 or More.	
	<2	2	4	8	16	32	64		Number.	Geometrical Mean of Reciprocal Titre.
New Guinea natives: adults	18	8	13	8	1	2	0	50	32	4·7
Blood donors in New South Wales, aged 18 to 59 years	23	9	7	17	21	3	0	80	57	8·2

specimens containing detectable amounts of hæmagglutinating antibody, calculated from the bench titres, were 0·036 unit per millilitre and 0·025 unit per millilitre, respectively (Tables IA and IB).

For comparison, the same tables also show results of diphtheria antitoxin estimations on 65 cord sera in Melbourne (Fisher, 1952) and on 2000 cord sera in London (Glenny, Barr, Billings and Butler, 1955). In the two series, the proportions of sera containing detectable amounts of hæmagglutinating antibody, or equivalent amounts of diphtheria antitoxin—35 out of 65 (54%) and 971 out of 2000 (47%) respectively—were of the same order as those present in the New South Wales donors, but much lower than those found in the specimens from New Guinea. On the other hand, the distribution of the titres in the cord sera, in the range where the antibody content was defined, resembled the distribution in the series from New Guinea, and the peak was appreciably higher than in the sera from New South Wales.

The intradermal toxin neutralization tests were made in rabbits on five pools prepared from a total of 25 specimens of New Guinea serum, the specimens being grouped for pooling according to their titres in the bench test. The antitoxin contents of the pools determined in the rabbit skin corresponded to antitoxin contents calculated from the titres determined *in vitro* (Table II).

The Presence of Antibody to H. Pertussis.

The pertussis antihæmagglutinin content in the sera of blood donoors from New South Wales was somewhat higher than in the specimens from New Guinea. In the former series, 57 specimens out of 80 (71%) contained detectable amounts of antibody, as against 32 out of 50 (64%) in the latter. The mean reciprocal titres, among the specimens with sufficient antibody to react in the test, were 8·2 and 4·7 respectively (Table III).

Discussion.

Faucial diphtheria has not been reported in the area of New Guinea where the sera tested in this series were collected. It has been stated to be uncommon among indigenous children in the tropics; however, cutaneous diphtheria has been reported to be of common occurrence, and has been surmised to be the cause of the very high percentage of negative results observed in Schick tests performed on some native children (Keizer, Fergusson, Lapierre and Monteny, 1952). More recently investigators from New Zealand (Bacon and Marples, 1955; Marples, 1955), working in Western Samoa, found a high rate of conversion to the Schick test in young children, and they isolated *C. diphtheriæ* with considerable frequency from different types of skin lesions in the same group, though only occasionally from the throat. It seems feasible that the presence of diphtheria antitoxin in the specimens of serum from New Guinea was due to skin infections harbouring *C. diphtheriæ*.

The proportion of New Guinea natives who, judging by the concentration of diphtheria antitoxin in their serum, could be expected to be non-reactors to the Schick test, was similar to the proportion found by the authors quoted above (Keizer *et alii*; Bacon and Marples; Marples). It is interesting to note that it was significantly higher than the proportion of adults with similar diphtheria antitoxin levels in New South Wales, and than the proportion of mothers in Melbourne and London, in previously reported series, as judged by tests made on cord sera; cord sera have been shown to be somewhat richer in diphtheria antitoxin than the corresponding maternal sera (Barr, Glenny and Randall, 1949). The explanation of the difference between New Guinea highlanders and Europeans, with respect to serum diphtheria antitoxin level, must lie in the differences in the epidemiology and bacteriology of diphtheria infection between communities living in tropical and temperate climates. While cutaneous diphtheria is uncommon among whites in the temperate zone, its occurrence was found to be not unusual in white military personnel in the tropics (Liebow, MacLean, Bumstead and Welt, 1946; Riddell, 1950).

Pertussis, diagnosed clinically, and causing considerable mortality, is known to have occurred recently in the Wabag area (Macintosh, 1955). The presence of the specific antibody in the sera from New Guinea may be associated with this recent outbreak.

Summary.

Diphtheria antitoxin was titrated in the sera of 43 adult males obtained from the Wabag area of New Guinea. All but three specimens contained at least 0·008 unit per millilitre. Of 50 specimens of a different series, obtained in the same area, antibody to *H. pertussis* (antihæmagglutinin) was detected in 18.

By comparison, of 80 specimens obtained from blood donors in New South Wales, diphtheria antitoxin amounting to 0·008 unit per millilitre or more was present in 45, and detectable quantities of pertussis antihæmagglutinin were present in 57.

Among the specimens containing not less than 0·008 unit of diphtheria antitoxin per millilitre, the mean antitoxin content in the New Guinea series was higher than in the New South Wales series. Among the sera showing detectable amounts of pertussis antihæmagglutinin, the mean antibody titre was slightly higher in the New South Wales series than in the New Guinea series.

The likely causes for the presence in the serum of the New Guinea natives of the antibodies referred to are discussed.

Acknowledgements.

The writer is indebted to Dr. R. J. Walsh and Dr. H. K. Ward for suggesting this survey, for supplying the sera, and for comments.

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PEPSINIZED GRASS POLLEN IN THE TREATMENT OF HAY FEVER.[1]

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THE use of pepsinized rye grass pollen given by intradermal injection was reported on favourably by Piper (1955*a*).

It was therefore decided to carry out a field trial of this material on a fairly large scale. Volunteers were obtained from three large industrial establishments near Adelaide. These were the Long Range Weapons Establishment at Salisbury, General Motors-Holden's, and the British Tube Mills in the north-western suburbs. These places were chosen because of their excellent casualty buildings and because the pollen exposure could reasonably be expected to be high. This applied particularly to Salisbury. No actual pollen counts were performed.

METHODS USED IN TRIAL.

There were nearly 300 volunteers. It was possible to treat and assess 253. Many of the remainder were treated, but were not included if a significant assessment of their result could not be made, or if pre-seasonal treatment within the last few years had been given.

An additional 16 patients were given histamine alone by the same method using the "blind trial" technique.

Interviews and Skin Testing.

All patients were personally interviewed. A brief history was obtained, particular note being taken of the duration and seasonal variation in symptoms, the presence of other allergic conditions, previous treatment, and recent environmental changes.

Scratch tests were performed in every case with the pollens of rye grass, flowers, couch grass, plantain, wheat and oat, and with the domestic allergens house dust, feathers and kapok.

On the history and the results of these skin tests, it was possible to divide the patients into the following four main groups: (i) grass pollenosis, with symptoms occurring only in the spring up to December; (ii) mixed pollenosis, in which symptoms occurred throughout the summer up to March or April; (iii) complicated allergy with perennial symptoms and a spring exacerbation; (iv) perennial rhinitis with no spring exacerbation. These are shown in Table I.

In addition, an attempt was made at the time of the initial interview to classify the severity of symptoms. These were grouped under three headings: (i) major disability, in which symptoms were sometimes sufficiently severe during the season to cause absence from work or serious interference with work; (ii) moderate disability, in which symptoms were sometimes severe enough to interfere appreciably with work; (iii) nuisance, in which minor symptoms only were experienced. These figures relating to the severity of symptoms are shown in Table II. They do not include those eight cases in which no spring exacerbation occurred.

[1] Read at a meeting of the Australian Society of Allergists on August 18, 1955, at Sydney.

TABLE I.
Seasonal Grouping.

Type of Allergy.	Number of Cases.
Grass pollinosis	95
Mixed pollinosis	86
Complicated allergy	64
Perennial rhinitis with no spring exacerbation	8
Total	253

Administration of Vaccine.

The aim in treatment was to produce five "adequate" reactions by intradermal injection into the skin of the volar aspect of the forearm. The injections were given twice a week. An "adequate" reaction was considered to be a weal one to two centimetres in diameter.

The majority of patients received no other injections. A few were given booster doses at the end of a month when all were reviewed, and a few received only three or four injections.

TABLE II.
Severity of Symptoms.

Degree.	Number of Cases.
Major disability	58
Minor disability	79
Nuisance	108
Total	245

Two solutions were used. The first solution given was one-tenth the concentration of the other. They were designated A/10 and A strength solutions. The initial dose was always 0·01 cubic centimetre of the A/10 solution (equivalent to 100 Noon units of rye grass pollen). Further doses were calculated from the size of the initial reaction. The maximum dose given at any one time was 0·1 cubic centimetre of A strength solution, which was equivalent to 10,000 Noon units of pollen prior to protein digestion.

After each injection, the skin reaction took up to fifteen or twenty minutes to reach its maximum size, and all patients were kept under supervision during this waiting period. This was in order to record their local skin reaction and so estimate the next dose, and also to watch for general systemic reactions. Seventeen such reactions were noted (approximately 1% of the numbers of injections). The reactions usually started with flushing of the face and a short cough, followed by initiation or exacerbation of hay fever symptoms. A few subjects developed bronchospasm, and one patient had rather alarming respiratory obstruction, cyanosis and abdominal pain. Rapid relief was given by the intravenous injection of "Anthisan", the subcutaneous injection of adrenaline and the application of a tourniquet. In some of the milder cases the subjects responded to the oral administration of "Neo-epinine".

No general reactions were observed which did not manifest themselves within twenty minutes of the injection. Severe reactions could usually be anticipated when there

was rapid spread of the weal with accompanying pseudopodia formation.

Some increase in size of the local reaction was common during the next twenty-four hours. The weal faded, to be replaced by a subcutaneous induration, sometimes quite extensive, and taking several days to disappear. It was not particularly painful. These were called local delayed reactions.

Preparation of Vaccine.

Full details of the method of preparation of the vaccine are given by Piper (1956).

Recording of Results.

Local skin reactions were classified according to the size of the weal, as follows: "+++", "++", "+" or "minimal". A "+++" reaction was a weal of about two centimetres in diameter, "++" 1·5 centimetres, and "+" 1·0 centimetre. The weal size was at first carefully measured at the end of the waiting period, but it soon became possible to assess these reasonably accurately without formal measurement. The accompanying flare was also roughly measured, but was not used in the final assessment of results.

The general state of symptoms was noted at each interview during the course, together with any local delayed reactions from the previous injection.

The majority of patients were interviewed again at the end of a month after the completion of treatment, and at this time, as has been indicated, a few booster doses of the preparation were given if symptoms were severe. If an interview was not possible, a *questionnaire* was sent out.

Two to three months after completion of treatment, all patients were sent a *questionnaire* (distributed in December and January).

RESULTS.

Crude Totals.

The cases were divided into five groups for assessment of the degree of relief. If "significant" relief is taken to include all cases in which 50% relief or more was claimed, it is seen that 177 patients fall into this category—that is, 72%. Only 11 patients of this group also took antihistamines, usually sporadically and not in a regular dosage.

TABLE III.
Relief Obtained.

Percentage of Relief.	Number of Cases.	Percentage.
90 to 100 ..	28	11·4 } 72·0
75	75	30·6 }
50	74	30·1 }
25	25	10·2
Nil	43	17·7
Total ..	245	

Seasonal Grouping.

Subdivision of the cases into the seasonal groups as shown in Table I produced the following results. It will be seen that significant relief was obtained in the grass pollenosis groups by 75%, in the mixed pollenosis groups by 74%, and in the complicated allergy group by 57%. Of the small groups of patients with no spring exacerbation of symptoms, 37% claimed adequate relief.

Response to Degree of Reaction.

An attempt was made to correlate the degree of local skin reactions and of general reactions with the degree of relief claimed. This is shown in Table V. When these figures were analysed, it was possible to demonstrate the average number of "+", "++" or "+++" reactions per case in each of the five groups. The only conclusion from such figures is that the larger skin reactions tend to occur slightly more frequently in patients who claim a poor result. There was no significant difference in the degree of relief obtained by those with local delayed reactions. These, together with the percentage of general systemic reactions in the five groups, are shown in Table VII. There was a significantly higher proportion of patients who had general reactions in the completely or almost completely relieved group.

TABLE IV.
Relief Obtained in Various Types of Allergy.

Percentage of Relief.	Percentage of Subjects Relieved.			
	Grass Pollinosis.	Mixed Pollinosis.	Complicated Allergy.	Perennial Rhinitis.
90 to 100	17 } 75	8 } 74	3 } 57	— } 37
75	28 }	29 }	18 }	2 }
50	26 }	28 }	20 }	1 }
25	10	6	9	1
Nil	14	15	14	4
Total ..	95	86	64	8

TABLE V.
Skin Reactions.

Percentage of Relief.	Degree and Type of Reactions.					
	+	++	+++	Local Delayed Reactions.	General Reactions.	Total.
90 to 100	29	79	36	7	5	28
75	85	221	110	19	3	75
50	96	237	104	29	7	74
25	31	84	39	7	2	25
Nil	59	137	69	13	—	43

Dosage.

The dosage was very variable, ranging from only a few hundred to 70,000 Noon units. There was no correlation between the dosage and relief claimed.

TABLE VI.
Average Results for One Case.

Percentage of Relief.	+	++	+++
90 to 100	1·04	2·82	1·28
75	1·13	2·95	1·47
50	1·30	3·20	1·40
25	1·24	3·36	1·56
Nil	1·37	3·19	1·60

Duration of Relief.

The majority of patients said that their relief lasted up to the last review. This was several months after the end of treatment. Eight patients said they experienced relief for only about one month, nine for several weeks, and six for only a few days. Almost all patients who claimed relief said this began during the course of injections.

Comparison of Most Severe and Least Severe Cases.

It was thought that a comparison of the most severe and the least severe cases might prove of some value owing to the unreliability of the patient's assessment of symptoms in a condition such as hay fever. However, there was very little difference between the two groups, significant relief being claimed by 67% in the most severe group, and by 73% in the least severe group.

Pre-Symptomatic and Post-Symptomatic Treatment.

The effect of treatment begun prior to any symptoms and that given after symptoms had appeared was assessed in the grass pollenosis and mixed pollenosis groups. This showed that in the grass pollenosis group, pre-symptomatic treatment gave a rather better result; 84% of subjects claimed significant relief against 63% of those treated after symptoms had begun. There was no significant difference in the mixed pollenosis group. More than twice as many patients were treated after symptoms had begun.

TABLE VII.
Relation Between Skin Sensitivity and Clinical Response.

Percentage of Relief.	Local Delayed Reactions.	General Reactions.
90 to 100	25%	18%
75	25%	4%
50	39%	10%
25	28%	8%
Nil	30%	—

Increase of Tolerance Against Injections.

There was undoubtedly a distinct tendency for a diminution in the size of the reactions to occur during treatment. However, as the number of injections given to each person was not great, it was not possible to assess this accurately, and in many cases it did not occur at all. There was no apparent relation between a "wearing out" of the reaction and an improvement in symptoms.

Complete Failures and Patients Possibly Made Worse.

There were 43 cases in the group of complete failures, equally divided among the three main types of allergy. There was no significant difference in dosage or degree of reaction between these subjects and those who claimed some relief. Nine patients stated that they had worse symptoms this year than in previous years, and several of these thought injections produced an exacerbation of symptoms.

Perennial Rhinitis.

It was decided to treat eight patients in this group who offered themselves to see if a similar response could be obtained. As can be seen from Table IV, only 37% of this small group claimed significant relief.

Over 60% of the patients who gave skin reactions showed a "++" reaction, and there were "+++" reactions in spite of the fact that there was no apparent spring exacerbation of symptoms, and scratch tests to grass pollen gave largely negative results.

Asthma and Hay Fever.

There were 30 patients in the asthma and hay fever series who admitted to definite asthmatic symptoms, usually with severe nasal symptoms. Twenty patients said that they had experienced some relief of their asthma as well as their hay fever, and of these 20 patients seven said that they had had complete relief from their usual asthmatic symptoms during the season. The remaining 10 said their asthma was unrelieved, and two of these said that it was worse than usual.

Histamine Series.

Towards the end of the trial, 16 patients received intradermal injections of histamine by the "blind trial" technique. A solution had been prepared to produce a weal about a similar average size to that produced by the pollen, and the same number of injections were given, the operator not knowing which solution he was giving. When the same criteria were used for assessing significant relief, 13 out of the 16 said they obtained this relief, three stating that it was almost complete. Of the six patients who stated that they experienced 50% relief, three said that it lasted for only a few weeks. There were no local delayed or general reactions.

Discussion.

The main purpose of this investigation was to determine whether this intraseasonal method provided significant symptomatic relief in allergic rhinitis, and if so, to seek some information as to the mode of action.

On the basis of the patients' own assessment it transpired that of the total series of 253 subjects, 177 or 72% claimed to have benefited appreciably (Table III). This observation means very little by itself, since this is a variable disease, the method of assessment was subjective, and psychological factors may have had a significant effect. If psychological factors had operated, it is likely that the effect would have been independent of the type of allergy. When the total series is so divided, as in Table IV, it can be seen that benefit from treatment was greatest in the cases preclassified as grass and mixed pollenosis (75%), less in the complicated allergy cases (57%), and least in cases of perennial rhinitis (37%). Since the vaccine was prepared from grass pollen, these findings do support the belief that this method was achieving some specific desensitization.

TABLE VIII.
Histamine Series.

Percentage of Relief.	Grass Pollinosis.	Mixed Pollinosis.	Complicated Allergy.
90 to 100	1	—	2
75	2	2	—
50	1	2	3
25	1	2	—
Nil	—	—	—
Total ..	5	6	5

However, there was very little evidence of any correlation between skin sensitivity, as judged by reaction to the injected material, and the clinical response (Tables V and VII). There was, in fact, rather a remarkably uniform incidence of degree of skin reaction and of local delayed reaction throughout the groups graded as to clinical response. A significantly larger number of general reactions occurred in those patients who subsequently claimed complete relief. This provides no information as to the specificity of the material, since relief may follow a systemic reaction provoked by any means. A non-specific action of the injected material is perhaps suggested by the findings shown in Table VI, in which the skin reactions in response to the injections of the course have been averaged in each clinical group. The skin reactions were as severe in the patients who claimed little or no benefit from the injections, if not more so. A small control series in which histamine was substituted for the pepsinized pollen material produced the somewhat disconcerting result that over 75% of the subjects claimed significant relief. It is difficult to believe that four or five intradermal injections of histamine would achieve true histamine desensitization, and the explanation may well be that this inadequate control group was treated too late in the season, and that natural subsidence of the hay fever was imminent in many of the subjects.

Many different schemes of pre-seasonal desensitization in hay fever have been advocated in which varying dosage, numbers of injections and timing of injections have been employed. It is therefore a matter of some surprise how similar are the reported results of treatment (Alexander, 1952). Much the same results are reported from the use of antihistamine drugs in the co-seasonal treatment of the disease (Mulligan, 1954; Arbesman *et alii*, 1947). By all forms of treatment about 80% of subjects are satisfactorily relieved and about 20% gain little or no benefit.

The fact that the method reported here has also apparently produced this common result, when applied intraseasonally and involving only a few injections, certainly indicates that the material and the method deserve

further study. This trial has not produced conclusive evidence of the value of this material or of its action, but further study is certainly indicated.

SUMMARY.

A method of intraseasonal desensitization in hay fever, by means of a pepsinized pollen preparation, has been submitted to simple clinical trial.

Two hundred and fifty-three volunteer subjects were treated, and 72% claimed to have obtained significant relief, while 28% derived little or no benefit.

There was no conclusive evidence as to whether the desensitization was specific or non-specific. It is considered that the material and the method warrant further study.

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PULMONARY TUBERCULOSIS: SOME ASPECTS OF SURGICAL TREATMENT IN PATIENTS DISCOVERED BY MASS X-RAY SURVEYS.

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> The sooner the cure of consumption is begun, the better it usually succeeds; and from hence this Distemper especially proves fatal, because the Physician is consulted when 'tis too late. . . .
>
> RICHARD MORTON: "Phthisiologia: or a Treatise of Consumption", London, 1689.

UNTIL the early years of this century, tuberculosis was diagnosed only after it was well advanced. Cough, hæmoptysis, fever, sweats and loss of weight made up the picture seen and taught to students up to recent times. Volumes have been written on the detection, diagnosis, treatment and supervision of the tuberculosis sufferer. As a result of the wider application of radiology, it became apparent that the disease could be present to an appreciable degree in the absence of symptoms.

Wunderly (1955), opening the discussion on "Mass Surveys for Pulmonary Tuberculosis" at the Australasian Medical Congress (British Medical Association), said that community-wide X-ray surveys still remained the best screening method leading to the detection of pulmonary tuberculosis amongst the apparently well. The expense of such an undertaking has been considered justifiable, since each active case discovered and rendered inactive would be one less source of infection within the community. From a surgical point of view, these surveys are well justified, as will be shown. In the epidemiological attack on tuberculosis these surveys are important, although some are of the opinion that investigation of selected groups would show a higher detection rate. It is known, for instance, that amongst patients referred by private practitioners the incidence is at least ten times as high as amongst the apparently healthy. Likewise, the incidence is higher in the city areas than in the country areas, whilst the highest incidence of all is found in the congested suburbs.

As a part of the Commonwealth Anti-Tuberculosis Campaign, X-ray examinations of the chests of the whole population aged over fourteen years were undertaken. In New South Wales surveys had been carried out previously on a voluntary basis. However, when compulsory attendance was introduced, a large number of infectious patients was brought to light (Rubinstein, 1956). As in other countries, most of the patients so discovered were in the less advanced and most treatable phase of the disease.

By early detection and treatment, mass case-finding programmes have made a considerable contribution to the recent decline in the tuberculosis mortality. In New South Wales during the past five years, surveys have been responsible for an increase of 19% in notifications of pulmonary tuberculosis, with a corresponding decrease in mortality. Initially this put a severe strain on sanatorium and chest hospital accommodation. Recently, this trend has been reversed, so that now there is practically no waiting for sanatorium accommodation.

An important contribution to the availability of sanatorium beds has been brought about by the wider use of surgery in the treatment of pulmonary tuberculosis. This has shortened the length of stay in hospital, a fact most noticeable in those cases discovered by mass surveys before symptoms appeared.

Godby (1953) described how he converted Bodington Chest Hospital, which before had been run on the old sanatorium lines, into an institution offering every type of treatment for pulmonary tuberculosis. He reported that in the first year after the conversion, 53 patients had had 100 major chest operations without a death. In the same hospital with two surgeons, the number of surgical patients has now increased to 436, on whom 577 operations have been performed, with an overall operative mortality rate of 1·7%—that is, whilst the patients were still in hospital, a minimum of three months. Of these 436 patients, 183 were discovered to have tuberculosis by mass X-ray surveys. The others were referred by general hospitals, private practitioners and chest clinics. Many others found to have abnormalities by mass X-ray surveys were referred to other thoracic units, where they had surgical treatment for tuberculous lesions.

These 183 patients were all submitted to surgical treatment after they had had an adequate trial of bed-rest and combined chemotherapy. Eighty-eight were operated on by myself and the rest by Dr. Ian Monk.

The age and sex distribution of these patients is shown in Table I. As can be seen, 75% of the surgery was carried out in the age group twenty to forty years.

TABLE I.

Age Group. (Years.)	Males.	Females.	Totals.
10 to 19	5	8	13
20 to 29	47	40	87
30 to 39	27	23	50
40 to 49	17	10	27
50 to 59	6	—	6
Totals	102	81	183

Table II shows the number of patients from the two sources of discovery submitted to the various operative procedures. The extent of operation required is an indication of the amount of disease present. There were five cases of bilateral disease in the mass survey group and 12 cases of bilateral disease in the other group.

As has been stated above, the over-all operative mortality rate for the combined series was 1·7%. There were nine deaths in the group brought to diagnosis from sources other than mass X-ray surveys, and only one in the group from mass surveys. This latter followed a pneumonectomy and thoracoplasty, which by the extent of the disease alone could hardly be considered as an early case.

With regard to late deaths, so far in the mass survey group no patient (apart from the single operative death) has died since operation. However, in some cases the follow-up period is as yet not very long, the longest being

four and three-quarter years and the shortest nine months. There have been five late deaths in the non-survey group in patients who had undergone operation. Only one of these has been due to a recurrence of tuberculosis.

TABLE II.

Nature of Operation.	Number of Operations.	
	Mass Surveys.	Other Sources.
Thoracoplasty (patients)	22 } 13%	89 } 37%
Extraperiosteal plombage	3 }	9 }
Pneumonectomy	3	16
Lobectomy *plus* segmental resection	19	26
Lobectomy	61	66
Resection of more than one segment	42 } 42%	41 } 22%
Resection of one segment	38 }	18 }
Totals	188	265

From Table II it is evident that surgical collapse therapy as a definitive operation is being undertaken less and less. Only 13% of the mass survey group had thoracoplasty or plombage as compared with 37% in the other group. This has been brought about by the wider use of chemotherapy, the earlier discovery of cases of tuberculosis, and the greater safety of resection techniques.

Thoracoplasty is performed as a staged operation for upper lobe disease. The diseased area of lung is detached from the chest wall to put it at rest so that it can benefit from the chemotherapy, and any cavitation can be closed, the area finally becoming fibrosed and even calcified. It is performed in stages, because the removal of several ribs in one operation may give rise to distress from pafadoxical respiration, even to the extent of producing death from cardio-respiratory embarrassment.

As may be seen from Table II, plombage was performed on only three patients from the mass survey group and on nine from the other group. Since it involves the introduction of a foreign material into the tissues of the body, it is not greatly favoured, and is reserved only for "poor risk" patients with upper lobe cavitation, whose pulmonary function and cardio-respiratory reserve have been taxed over a long period of illness, and for whom a staged thoracoplasty or resection would be too great a risk to life. The principle of the operation is to relax the lung as in thoracoplasty, without removal of ribs and in one operation, the plomb being inserted to maintain the lung in its new relaxed position; it also prevents any paradoxical respiration.

From the whole series it is seen that resection has become the procedure of choice. This was applied in 87% of the mass survey group and in 63% of the other group. It is interesting to note that almost twice as many segmental resections were performed in the mass survey group as among the patients discovered from other sources.

The nature of the resection operations is self-explanatory; but it may be added that all the pneumonectomies were combined with thoracoplasty, as were 50% of lobectomies *plus* segmental resection and 8% of lobectomies only. A thoracoplasty in these circumstances consists of removal of a number of ribs to allow the chest wall to fall in; this lessens the space which the portion of the lung left behind has to occupy. This is to prevent over-distension of the lung tissue which is left, as this would impair its function, as well as carrying the risk of fistula and infection in some cases.

Indications for Surgical Treatment.

The following is the list of indications for surgical treatment which has been followed.

1. Persistent cavitation despite chemotherapy. The nature of the operative procedure will vary with the site of cavitation.

2. Residual lesion after chemotherapy. A solid lesion one or more centimetres in diameter is recommended for resection. This figure may seem arbitrary, but it is generally agreed that in the majority of cases such a lesion will fail to resolve by continuation of the chemotherapy alone.

3. Failed collapse therapy. This may have been pneumothorax, pneumoperitoneum, thoracoplasty or plombage. A resection may have been too hazardous as primary treatment; but after thoracoplasty, which has failed to achieve the desired result, it may nevertheless have rendered the patient able to withstand resection as a secondary procedure. In the earlier days of resection some surgeons deliberately performed a thoracoplasty as a first stage before proceeding to resection.

4. Solid tuberculous lesion, "tuberculoma". This can be a progressive primary lesion, a circumscribed area of caseous pneumonia, a blocked cavity or a tuberculous abscess distal to an obstructed lesser bronchus.

5. Bronchial pyelocele. This is one result of an obstruction to a medium-sized bronchus. This obstruction may be by pressure from without the lumen, due to a caseous mass or lymph node, or may follow bronchial disease resulting in a stricture beyond which caseous material collects.

6. Destroyed lung. This may arise in two ways. Firstly, bronchial disease with subsequent stricture, atelectasis, secondary infection and bronchiectasis can result in destruction. Secondly, advancing tuberculous infection may cause a great amount of destruction to lung tissue before the commencement of healing.

7. Lower lobe disease. Cavities in this region appeared to have been amenable to paralysis of the hemidiaphragm with pneumoperitoneum. However, those situated in the apical segment did not. Many of those in the subapex and basal segments which did respond initially, reopened later.

8. Lesions situated anteriorly. Persistent lesions confined to the anterior segments of the upper lobe, the lingula, or the middle lobe are better resected, as they do not respond well to thoracoplasty.

9. Tuberculous empyema. It has only been since the availability of the anti-tuberculosis drugs and the more frequent application of resection that a formal attack on tuberculous empyemata has been undertaken. The practice now when possible is to excise the empyema, the diseased underlying lung from which the empyema originated, and decorticate the remaining uninvolved lung—a formidable programme, but with gratifying results in most cases.

Though surgical collapse procedures have long been in use, it is only of recent years that resection has been carried out extensively. This has come about by a better understanding of the pathology and bacteriology of the disease. The interpretations of Medlar (1955), of Canetti (1955) and of others have been studied. In addition to such authoritative sources, there are some conclusions to be drawn from all the material removed at resection from the above-mentioned patients, all of which has been subjected to macroscopic and microscopic examination.

From these studies an attempt has been made to try to assess from the pathological findings how long after operation it will be advisable to continue with chemotherapy. At present, though this is perhaps being over-cautious, if examination of the specimen shows that the lesion is still progressive, a long course is maintained, lasting up to eighteen months from the date of operation. During the latter months of such a course PAS and "Isoniazid" are given, to enable the patient to work whilst still under treatment. Since all the patients have had chemotherapy before operation, most of the lesions are reported as being regressive, and when this is so it is usual to cease treatment within a year of operation.

Bacteriology.

Of the group of 183 patients found by mass survey to have tuberculosis, in all except 7% the diagnosis was confirmed bacteriologically before operation. In 132 cases (72%) tubercle bacilli were grown from the sputum, and in 38 (21%) bacilli were grown on culture from the gastric contents.

A bacteriological examination of the resected specimens was not undertaken, but studies made by others have revealed some interesting facts. Canetti (1955) found that the specimens from patients with residual cavitation after what was considered to be adequate chemotherapy still contained viable tubercle bacilli. The only exceptions to this were the patients in whom the remaining cavity had a smooth fibrotic wall. The closed caseous lesions which include what are called blocked cavities, as well as the caseous lesions which have never cavitated, may also contain viable bacilli, demonstrable by Ziel-Neilsen staining. Medlar (1955) held that in the main, bacilli from closed lesions failed to grow on culture and would not produce disease in guinea-pigs—an opinion not generally held. However, he was able to grow bacilli from every specimen in which there was a still patent cavity, even after the patients had had three years of chemotherapy.

This non-viability of bacilli in closed lesions was observed even before the days of chemotherapy; but it has not yet been proved that this is a permanent state. In fact, work by Hobby and co-workers (1954) suggests that with special cultural methods these bacilli will grow, so that such lesions must be considered potentially noxious and capable of flaring into activity. Auerbach (1955) found viable bacilli in 73% of resected specimens from patients who had had combined chemotherapy for from four to eight months. He found no significant difference whether the streptomycin had been given daily or twice weekly. It is generally held that with adequate cultural facilities, viable tubercle bacilli can be demonstrated in caseous or cavitated lesions which persist after chemotherapy.

Pathology.

As elsewhere, pathological examination has been extensively carried out at Bodington Chest Hospital. All the resection specimens were examined macroscopically alongside the X-ray films and tomograms. It is hoped that it may be possible in time to read the X-ray films with a pathologist's eye, and to anticipate with some degree of accuracy the nature and probable behaviour of the lesion. If this information is coupled with the findings in those specimens which have been removed, it may become possible to assess radiologically which lesion is likely to respond to a chemotherapeutic regime alone.

In the group of patients discovered by mass survey, on examination of sections 67% of the resected specimens showed caseation alone, 8% showed predominantly cavitation and 25% showed both caseation and cavitation. Bronchial disease was demonstrated in 13% of the series. After operation, only two patients have been found to have tubercle bacilli in gastric contents, and subsequently they could not be found.

The effects of chemotherapy have been observed, as well as the part it has played in the natural history of the disease. From Canetti's work (1955) it is evident that maximum benefit is derived from chemotherapy with the disease either in the stage of exudative alveolitis or in that of perifocal inflammation. In fact, in these stages, chemotherapy can be looked upon as a cure. However, when caseation has taken place, the effectiveness of chemotherapy is much reduced, and if it is persisted with, it will produce some cases which can be considered as "chemotherapy failures". If a caseous area is small and recent enough, chemotherapy may reduce it to a fibrous scar; but in the majority of such cases the best that can be hoped for is a resolution of the surrounding exudative disease with residual nodules incapable of further healing.

A stage further than caseation is cavity formation. Here the response to chemotherapy is variable. A recent cavity may react quite dramatically, rapidly becoming smaller in size and leaving only a scar, or it may only be reduced in size. The lumen can become blocked with necrotic material, and so it becomes a blocked cavity, an entity which is seen more often since the use of chemotherapy. When the cavitation is of longer duration, chemotherapy may render the contents sterile, and when it is surrounded by firm fibrosis little more than this can be hoped for. Canetti considers the blocked cavity a stage towards healing; but experience has shown that this type of lesion is prone to relapse at some future date, even after a prolonged course of chemotherapy and apparent inactivity. Ideally, the healing of a cavity follows the approximation of its walls after extrusion of the contents. For this reason, though a blocked cavity may lead to healing, from the point of view of stability it must be considered in the same category as the solid lesion with an unpredictable future.

In a personal communication McGovern (1956), from examination of the present series of resection specimens, has noted the tendency towards healing of the tuberculous process in the lymphatics leading from the caseous lesion. This takes place whether the main lesion is progressive or not. This factor is most important in segmental resections, in which the paths of these lymphatics are most likely to be crossed. The fact that one rarely sees a new focus of disease after resection suggests that the plane of cleavage has been across inactive if not non-existent disease. From study of the specimens it seems that the anti-tuberculosis drugs have not altered the mechanism of the disease, but have caused a general speeding up of the healing process. Thus a more rapid hyalinization of the margins of a caseous mass prevents liquefaction and cavity formation. The concurrent rapid healing of the lymphatic-spread tubercles prevents diffusion of new lesions which could act as secondary foci of dissemination.

On pathological grounds, McGovern advises resection for large lesions even though they are undergoing satisfactory regression. For the smaller persistent ones resection is advised, since the healing process at first accelerated later becomes slow.

"Tuberculoma."

The term "tuberculoma" has been accepted as a radiological description of a circumscribed lesion of tuberculous origin not less than one centimetre in diameter. It could be accounted for by a progressive primary focus, an area of caseous pneumonia, a blocked cavity or a tuberculous abscess distal to an obstructed lesser bronchus. The fate of these lesions is so far unpredictable, and it is thought that since this type of lesion is seen so frequently in mass X-ray surveys, a special mention is merited. Opinions still differ concerning the management of these lesions.

Campbell (1955) described a series of eight tuberculomata which disappeared spontaneously and without any evidence of activation of disease. He considered them to have been evacuated, and in fact in two of his cases a thin-walled space was evident for a while at the site of the earlier lesion. That they were evacuated without manifest disease is taken to be due to the fact that they had no viable bacilli in them at the time. He quotes various authorities as stating that, from extensive observations, they are of the opinion that as a general rule medical treatment is preferable to resection. Moyes (1951), one of the authorities quoted, reviewed a series of 41 patients with tuberculomata who, except for seven who underwent resection, were followed up for from three to fifteen years. Though there were no deaths, in half the cases it was clear that the lesion was unstable, and in fact 17 patients developed cavities or new areas of disease which responded to treatment. Many of Moyes's patients were followed in retrospect, and in the light of recent knowledge several of them would have been recommended for resection much earlier in their long history. Since such lesions are often detected in people without symptoms, are they to be permitted to carry on their lives as usual, or must they be advised to curb their activities? The stage has not yet been reached when it can be said on radiological grounds, even if the lesion is considered to be tuberculous, that it is not one which is about to spread, increase in size or cavitate. If the diagnosis of tuberculoma is established, conservative management can be advised, because if spread or breakdown occurs, chemotherapy in a sanatorium will control it. Though many of these lesions are tuberculous despite negative bacteriological findings once these patients have had their attention focused on an abnormality, it is unlikely that they will carry on with a care-free mind. Often, such lesions having been removed and proven to be tuberculous, the patient has returned to

his work in a short time free from anxiety about a subsequent breakdown.

The extensive amount of pathological and bacteriological work which has been and still is being carried out supports the practice of surgical resection of residual disease at the present time. Undoubtedly chemotherapy plays a big part, but too frequently relapses occur with this treatment alone.

Discussion.

There is another aspect of the mass survey tuberculosis case-finding programme worthy of comment. One might ask, what would have been the fate of these patients discovered by mass survey, had they not been brought to light when they were? It must be admitted that a few of them would probably have come to no harm. Their lesions would have healed or become non-infectious and they would have lived their allotted span without any trouble. In most of them, however, the disease would have progressed until such time as the patient became ill and sought medical advice. All the time his disease was progressing he would have been a source of infection to others. By then the advanced nature of the lesion would have necessitated prolonged therapy, after which the best that could have been achieved would have been chronic ill-health and incapacity for regular work. On the other hand, many of the patients in the foregoing series discovered by mass survey and submitted to operation have been back at work in their original jobs, still perhaps receiving chemotherapy, within a year of their initial mass survey X-ray examination. When these are compared with people who are left until they are ill before they come to treatment, it can be estimated very conservatively that at least one year is being saved per patient by the fact that their condition was diagnosed whilst they were asymptomatic.

Considering the surgical management of pulmonary tuberculosis as applied to the present-day occupants of sanatoria and chest hospitals, Keers (1955) grouped his patients in the following three categories, which could equally apply to the present series: Group I: cases in which there is some doubt as to whether operation will be required; Group II: cases in which operation is clearly indicated; Group III: cases in which operation would be desirable if the extent and distribution of the disease or the complicating factors did not render it impracticable or unduly hazardous.

It can be seen that nearly all the 183 patients discovered by mass survey, on whom operation was performed, fall into Groups I and II. Rubinstein (1956) has shown that mass surveys have revealed a large number of patients with advanced disease, of whom many by age alone are beyond even the Group III category. Likewise, the patients who come to diagnosis because of ill health or with significant symptoms also mainly fall into this category.

These three groups of patients vary in their management.

With Group I patients, a vigorous application of combined chemotherapy may produce a cure. If, after this, a lesion greater than one centimetre in diameter persists, the patient can then be considered as in Group II.

The management of Group II patients is relatively straightforward. The indications for operation have been given. With these in mind, it remains but to choose the optimum time and the best procedure for the individual patient.

Group III includes the patients who were seen so commonly in sanatoria before the advent of chemotherapy and mass X-ray surveys, and before surgical treatment was readily available. Such patients can still be found in sanatoria run on purely conservative treatment, where they have usually been for several years consecutively or with remissions. They present a problem of salvage from a surgical viewpoint, having usually been under treatment for a considerable length of time, and in many cases having had varying periods of the lesser collapse procedures. These are the patients who lend support to the consideration of early operation in the treatment of pulmonary tuberculosis. A study of them in retrospect, when this is possible, will show that many at one time had their disease in one lung only, where it had a lobar or even segmental distribution. As seen in Group III, they present a serious problem of assessment. The extent of lung damage by fibrosis, the presence of secondary infection, the state of the cardio-respiratory reserve from prolonged hospitalization, and their chances of mental and physical rehabilitation must all be taken into account. Many have tubercle bacilli resistant to one or all of the three main anti-tuberculosis drugs. It is this group of patients which gives rise to a lot of work. In some cases risks have to be taken. Since the outlook for these patients is otherwise hopeless, the effort and risk are justified.

Results vary with the number of "poor risk" patients accepted for operation. If a comparison is made between Group III patients and those whose disease has been diagnosed early and who have had early surgical treatment, the difference is most impressive. Forty-seven such patients are included in this combined series, only 18 of whom were found by mass survey. Though they have accounted for most of the operative mortality and morbidity due to the extensive surgery required on "poor risk" subjects, the results in the main have been most gratifying. In the cases diagnosed early, in which early resection has been carried out, the mortality and morbidity are almost negligible.

A further comparison of interest is the duration of disability in patients from the two sources of discovery. When the length of time is calculated from diagnosis, through the various lines of non-surgical treatment, to the decision for operation and then till the patient returns to work, it will be found to add up to several years for many of the patients from sources other than mass surveys. The average time from diagnosis to return to work for the 154 patients discovered by mass surveys who have so far returned to work, has been seventeen months.

In terms of cost, and with the exclusion of the value of lost man-hours of labour and therefore of earning capacity, each patient presumably saved a year of disability represents a saving to the Commonwealth exchequer. If the average tuberculosis allowance is taken at £7 per week, these patients (183) will have saved £64,050 in tuberculosis allowances. At a low estimate of £3 per day for hospital accommodation and treatment, an annual saving of £1100 per patient is involved. These 183 patients discovered by mass surveys thus represent a saving of £265,350. If full access to records had been available, this sum would have been much larger, since a number of survey-discovered patients have had surgical treatment at other hospitals.

Conclusions.

By early discovery of tuberculous subjects through mass X-ray survey and prompt treatment, valuable time is saved in hospitalization. Discomfort to patient and family, loss of time from work and expense to the community in pensions and hospital accommodation are minimized. Even more important from a public health point of view is the elimination of potential sources of infection amongst apparently healthy people. It is agreed that surgical treatment, especially excision, is most likely to overcome the tendency for tuberculosis to recur. It is generally felt that a combination of treatment by bed-rest, chemotherapy and surgery yields the greatest number of good results in the shortest time. To achieve this, early diagnosis is very important. This is a most essential factor when one considers that it is often the breadwinner who is affected, and that it is the community itself which has to provide for those suffering from pulmonary tuberculosis.

In the fifteenth century, Nicolò Machiavelli made the following statement about consumption:

> In the beginning it is easy to cure and difficult to understand, but when it has neither been discovered in due time nor treated upon a proper principle, it becomes easy to understand and difficult to cure.

With mass radiography to "understand", and chemotherapy and resection as the "proper principle", a cure can be effected. Properly conducted case-finding programmes, the use of chemotherapy and the practice of resection when indicated, can greatly contribute to the elimination of pulmonary tuberculosis.

Summary.[1]

1. Mass X-ray surveys in New South Wales have brought to light a large number of patients with active tuberculosis amongst apparently healthy people.

2. One hundred and eighty-three patients so discovered have been treated surgically. These are combined and compared with 253 tuberculosis patients discovered from other sources who have had surgical treatment in Bodington Chest Hospital with an overall operative mortality rate of 1·7%.

3. Of the survey-discovered patients, 87% were suitable for treatment by resection, as compared with 63% of the patients from other sources. The amount of surgery required in the survey patients was much less extensive than in the other group.

4. Bacteriological and pathological aspects of tuberculosis, as observed in resected specimens, are discussed, with special reference to tuberculoma.

5. Seventeen months is the average length of time from diagnosis to return to work for those patients discovered by mass surveys. In comparison, the duration of disability for patients whose illness was detected as a result of symptoms could involve several years.

6. An estimate of the economic saving is given amongst patients discovered by mass surveys and followed by surgical treatment when indicated.

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I wish to thank the President and Board of Directors of the Anti-Tuberculosis Association of New South Wales for permission to publish this article. Nearly all the mass survey patients and the majority of the non-survey patients have been discovered through this association, and the follow-up investigation of most of them has been carried out at its clinic. I am indebted to Dr. Ian Monk for permission to include his patients, and to Dr. N. R. Godby, medical superintendent, and the Australian Red Cross Society, the administrators, of Bodington Chest Hospital, where all the operations were performed. I am grateful to the numerous medical practitioners who readily responded to the follow-up *questionnaire*.

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[1] Since this paper was submitted for publication, another 40 patients discovered by mass X-ray surveys have had surgical treatment, as well as another 37 admitted to hospital from other sources.

Reports of Cases.

ANNULAR PANCREAS: A CASE REPORT.

By D. J. WURTH, F.R.C.S.,
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DURING the development of the gut in the embryo, failure of the ventral *Anlage* of the pancreas to rotate with the duodenum is thought to cause the condition of annular pancreas (Wakeley, 1951).

A ring of pancreatic tissue continuous with the main gland surrounds the second part of the duodenum. Duodenal obstruction causing symptoms such as nausea, vomiting and epigastric pain may be produced. The duodenum proximal to the annulus becomes dilated, often enormously, and there is sometimes an associated peptic ulcer.

Rarely is the diagnosis made pre-operatively, and most cases are found at post-mortem examination. Up to 1953 only 78 cases had been recorded, of which surgical treatment had been given in 28 (Wilson and Bushart, 1953).

Treatment consists of either excising the annulus or performing a short-circuit operation.

Clinical Record.

A female patient, aged thirty-nine years, had suffered from attacks of colicky epigastric pain since 1951. These lasted for two or three weeks and were accompanied by flatulence and relative constipation. The first attack had been followed by a brisk hæmatemesis and melæna; but subsequent barium meal X-ray examination revealed a dilated duodenal cap, but no peptic ulceration.

In January, 1955, I examined the patient during an attack. The only finding was the presence of an ill-defined, doughy, epigastric mass, which on palpation gave rise to much splashing and borborygmus. Barium meal X-ray examination again revealed great dilatation of the first part of the duodenum, an appearance suggestive of duodenal diverticulum.

At operation by Dr. B. T. Edye, in February, 1955, the proximal part of the duodenum was found dilated to the size of a sphere four inches in diameter. At the angle between the first and second parts there was a ring of pancreatic tissue a quarter of an inch thick, completely encircling the duodenum and obstructing it. The ring was divided and excised easily, and an anastomosis was made between the dilated first part of the duodenum and the collapsed second part. This was necessary, as the bowel wall appeared permanently constricted under the annulus. No peptic ulceration was found.

Convalescence was stormy, owing to swelling of the stoma; but the patient is now well a year and a half later, and free from attacks of obstruction.

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I wish to thank Dr. B. T. Edye, who performed the operation, and Dr. A. Sharland for the X-ray examinations.

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ACUTE PHLEGMONOUS CAECITIS: REPORT OF A CASE.

By TERENCE W. HORNE, M.B., B.S., F.R.A.C.S., AND DONALD CAM, M.B., B.S.,
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ACUTE PHLEGMONOUS CAECITIS is a not uncommon condition. In spite of this fact, it is safe to say that most text-books do not even mention the condition, and that many surgeons have never heard of it. The lesion is

important because perforation of the caecum may occur, giving rise to general peritonitis. For this reason, it is deemed advisable to publish this report of a new case in which perforation had occurred, and which is unusual in that there was coincidental acute appendicitis. This is the fourth example of this disease which has been found at the Royal Hobart Hospital during the past three years. Two of the previous cases have already been described (Horne, 1955).

Clinical Record.

Mr. A., aged fifty-four years, was admitted to the Royal Hobart Hospital on October 31, 1955, with a provisional diagnosis of acute appendicitis. For the previous two weeks he had experienced anorexia, nausea and a vague feeling of ill-health. Two days before his admission to hospital, while working on night shift, he had suddenly felt nauseated, and had then passed three loose bowel motions in four hours. He stayed in bed all the next day and did not go to work the next night. He felt much the same during the day prior to his admission to hospital, but at 3 a.m. the next day he suddenly developed a pain in the right lower quadrant of the abdomen. The pain was constant and gnawing in type, and did not radiate in any direction. It increased in intensity and became unbearable. He was admitted to hospital at 7 a.m. There had been no vomiting or further diarrhœa. The patient had had two previous attacks of similar but milder pain lasting for two days in 1953 and in April, 1955. He said that he had had a coronary occlusion fifteen years before.

On examination, the patient was flushed and lying still in bed. The temperature was 101° F., the pulse rate 90 per minute, and the respirations numbered 20 per minute. The blood pressure was 150 millimetres of mercury, systolic, and 90 millimetres of mercury, diastolic. The tongue was heavily coated and the breath was foul. Guarding was present in the abdominal wall, more pronounced in the lower part of the abdomen and especially on the right side. There was generalized abdominal tenderness, and this was exquisite in the right lower quadrant. Release tenderness was present over the whole abdomen, but was also most pronounced in the right lower quadrant. Rovsing's sign was present. No bowel sounds were heard on abdominal auscultation. Rectal examination elicited pronounced tenderness high up on the right side. Examination of the other systems revealed no abnormality.

A diagnosis of acute appendicitis with perforation was made, and immediate operation advised.

The abdomen was opened by a muscle-splitting incision in the right lower quadrant. Free fluid was present in the peritoneal cavity. The caecum was found to be generally inflamed, with a thickened and indurated area about one inch in diameter in the lateral wall. In the centre of the indurated area was a perforation about a third of an inch in diameter. The appendix was found to be retrocaecal and bound to the caecum. The tip of the appendix was gangrenous, and was embedded in the caecal wall about an inch from the indurated area. A portion of the caecal wall forming the margin of the perforation was excised for histological examination. The perforation was closed with a free omental graft, sutured loosely to the caecum, and the appendix was removed. The abdominal wall was closed in layers, and a drain was left down to the caecal area.

After operation the patient was treated with gastric suction, with the intravenous administration of fluids, and with "Terramycin", which was given intravenously at first and later by the intramuscular route. He had prolonged paralytic ileus and was not able to take fluids by mouth till the twelfth post-operative day. The drain was removed on the third day. No significant drainage occurred. On the ninth day he developed a mild chest infection, which subsided with the aid of physiotherapy.

On November 20 he was transferred to a convalescent hospital. On November 28 he was readmitted to hospital with thrombosis of the left femoral vein, which was treated with rest and anticoagulants. He was finally discharged from hospital on December 31, feeling well, and wearing an elastic stocking to control the œdema of the left leg.

The pathologist's report was as follows: "The piece of caecum shows necrotic and inflammatory changes. No malignancy seen."

The patient was examined as an out-patient on February 13, 1956. He said that he was very well and had no pain, his bowels were open regularly, and the swelling of the leg was adequately controlled by the elastic stocking. The wound was found to be well healed except for an area of slight weakness at the site of the drain. There was no abdominal tenderness.

Comment.

The coincidence of acute phlegmonous caecitis and acute appendicitis has not been, to our knowledge, previously reported.

Acute phlegmonous caecitis is thought to be a submucosal infection, probably originating from the bowel contents, possibly by inoculation with a foreign body. Similar lesions have been reported elsewhere in the gut and in the stomach. It is at least possible in this case that the primary lesion was the acute inflammation of the appendix, and that infection spread from the appendix to the caecal wall.

Various methods of dealing with the perforation have been advocated. These include exteriorization of the caecum, excision of the lesion with suture of the defect in the caecal wall, and closure of the perforation with sutures. Exteriorization of the caecum can be difficult, and the procedure opens up the retroperitoneal tissue plane to infection. Also there is a faecal fistula to be dealt with later. Excision of the lesion again may be very difficult, and in any case it seems superfluous to sacrifice even a small area of gut for a self-limiting disease. Suture of the perforation is difficult and uncertain because of the friability of the caecal wall, which will not hold sutures readily. This last consideration suggests the analogy of the ruptured peptic ulcer, in which omental graft, either free or otherwise, without closure of the perforation with sutures, has proved entirely successful. It is thought that, in the presence of peritoneal inflammation, the graft adheres rapidly—probably within twenty-four hours. The invariable paralytic ileus prevents significant spillage from the already empty gut, and drainage of the area is a safeguard should any leakage occur.

In conclusion, closure of the perforation with omentum is simple and seems to be effective.

Acknowledgements.

We are indebted to Dr. C. Duncan for the report on the biopsy.

Reference.

HORNE, T. W. (1955), "Acute Phlegmonous Caecitis—A Report of Two Cases", *Australian & New Zealand J. Surg.*, 14: 279.

Reviews.

Progress in Neurobiology: I. Neurochemistry. Edited by Saul R. Korey, M.D., and John I. Nurnberger, M.D., with 23 contributors; 1956. New York: Hoeber-Harper, Incorporated. 9" × 6", pp. 225, with illustrations. Price: $6.75.

A NEW series of symposia entitled "Progress in Neurobiology" is in process of production, and the first volume, "Neurochemistry", has recently appeared. This has sixteen articles on various aspects of neurochemistry by 24 contributors—neurologists and biochemists working in neurological departments. The articles cover a wide field, some being purely chemical, such as "The Cerebral Metabolic Rate in Children" and "The Relation of Cerebral Circulation and Metabolism to Mental Activity".

The book can be looked on as a useful summary of the present position regarding some aspects of the chemistry of the central nervous system. The knowledge presented does little towards an understanding of the mode of working of the central nervous system. The brain is notoriously a difficult organ to work with, and the results given in this

book can be looked upon mainly as a survey of the field to be examined with, at present, no definite conclusions. The psychiatrist, with sufficient knowledge of modern biochemistry, will find much of interest in this book, but the general medical reader will find it rather difficult and not very rewarding. To the research worker in this field the book is a mine of information.

Gullan's Theory and Practice of Nursing. By M. A. Gullan, revised by Marion E. Gould, D.N. Seventh Edition; 1956. London: H. K. Lewis and Company, Limited. 5¼" x 8¼", pp. 309, with illustrations. Price: 18s.

THE 1955 edition of this book includes details omitted from previous editions, such as "mask technique" and the importance of a dust-free atmosphere, in the chapter on surgical dressings, but still omits certain procedures and diagrams which would make the book much more useful as a reference book. Diagrams are still too few. Two important diagrams immediately coming to mind are those for tidal drainage and Wangensteen's apparatus.

With the growing importance in medicine of fluid and electrolyte balance, it is surprising that all mention of fluid balance charts is missing; similarly no mention is made of nursing duties in relation to the intravenous administration of fluids, and especially blood transfusions. Some of the space devoted to the nursing of typhoid fever patients and such obsolete nursing procedures as ice cradling, turpentine stupes and cupping, could well be utilized for more modern techniques.

The addition of the chapter on radiotherapy is an extra advantage and very necessary for nurses in training, but it could be more explicit. Nursing care is not mentioned in regard to patients with radium *in situ*.

The chapter on "Hints on Private Nursing" is excellent, and most helpful to the new graduate.

Sterilization methods given are very helpful for domiciliary nursing, but more details could be given of hospital routine, and of the use of modern high-pressure, high-speed methods of sterilization.

In the preface the author states that this edition is an extension of the sixth edition, but that "opportunity has been taken to make a few adjustments and minor nursing additions". All these greatly increase the value of the book. With the increasing demand for more highly skilled nursing techniques, and continual expansion of the trainee nurses' curriculum, much more could have been added.

As it stands today, the text is too far advanced for the junior nurse and not sufficiently detailed for the senior. Unfortunately with the new syllabus now in use in Australian training schools, the book would be of no great assistance as a reference book.

The Body Fluids: Basic Physiology and Practical Therapeutics. By J. Russell Elkington, M.D., and T. S. Danowski, M.D.; 1955. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 6", pp. 646, with many illustrations. Price: £5 10s.

FEW aspects of medicine have developed so rapidly as the study of the physiology of body fluids and electrolytes and particularly its therapeutic application. This is so clearly reflected in the contrast between the rather self-conscious reluctance of physicians five or six years ago to accept the new style of expression of electrolyte concentrations in terms of milliequivalents and the current universal usage of these units.

With the exception of the discovery of antibiotics, it is difficult to think of any aspect of medical management that has contributed so much to the relief of suffering and the saving of life. There is no field or specialty which is not concerned, and the subject provides a common meeting ground for physician, surgeon, anæsthetist, pædiatrician and pathologist. Even dermatologist and psychiatrist must be prepared to recognize the symptoms of fluid and electrolyte imbalance which may occur in their patients.

An extensive and thorough review such as this book provides will thus appeal to a very wide circle of readers, and it can be recommended with confidence. The subject is approached from every conceivable angle. The introduction includes discussion of the chemistry, dynamics, evolution and comparative physiology of body fluids as well as a description of the techniques of their study.

A long section on physiology follows; in fact it is rather too long. The arrangement is complex so that after a lengthy discussion of the effects of pathological processes—starvation, dehydration, diarrhœa *et cetera*—repetition occurs in the discussion on water, sodium, potassium excess, and then again on deficiencies of these substances. The wealth of information in this section is also rather marred by the inclusion and repetition of quite elementary observations—no doubt admirable for the junior student—but as the book constitutes a valuable reference work, this section could with advantage be pruned to bring it into line with the subsequent, more business-like clinical chapters.

The latter are clear, precise and complete and deal with renal, cardiac, gastro-intestinal and endocrine diseases, and also general pædiatric and surgical problems. Of particular value to the clinician are the final chapters on the practical procedures in fluid therapy—the calculation of deficits and requirements and the description of methods of administration.

The work is profusely illustrated by graphs and charts, which are sometimes not as clearly set out as they should be. The bibliography is voluminous, and the frequent appearance therein of the authors themselves testifies to their extensive experience and original contributions to the subject.

An Atlas of Regional Dermatology. By G. H. Percival, M.D., Ph.D., F.R.C.P.E., D.P.H., and T. C. Dodds, F.I.M.L.T., F.I.B.P., F.R.P.S.; 1955. Edinburgh and London: E. and S. Livingstone, Limited. 9¼" x 6", pp. 272, with 475 illustrations. Price: £5.

THIS work is a most worthy undertaking, which stresses again the great value of the coloured photograph in the study and teaching of skin diseases. However, it also emphasizes that such photographs can never be substitutes for the naked-eye examination of the lesion on the patient.

Coloured photography in diseases of the skin is rapidly improving in quality, but this book shows that the techniques are not yet by any means perfected. Certain colours are exaggerated, for example, pinks tend to be too pink, as seen in Figures 180, 181. The effort in clinical photography must be directed towards accurate reproduction, and where this cannot be assured, the particular photograph would be best omitted, for example, Figure 184 (*necrobiosis lipoidica*) is a bad photograph. The disease depicted could be anything. Likewise, Figures 186, 188, 191 (*epidermolysis bullosa*, dermatomyositis, *verruca vulgaris*) are not sufficiently precise to enable the disease to be accurately diagnosed. Blurred specimens (and there are a few) should be also omitted.

The method of regional arrangement is good and of value in teaching of differential diagnosis.

In Australia, progress in colour photography of skin diseases has been considerable over the past ten years. Many dermatologists possess their own collections of transparencies which are invaluable in teaching and revising. The great worth of this volume is in the teaching of undergraduates—of filling in the gaps in their knowledge. It is of value also to the specialist who may not often see such diseases as papulo-necrotic tuberculides (Figure 26). There is sufficient text to justify the specialist's perusal and on which to base his teaching.

The authors must be complimented on a useful and valuable teaching atlas, and, with a few exceptions, on the quality of their photographic reproductions.

Practical Urology: Case Comments and Late Results. By Alex E. Roche, M.A., M.D., M.Ch. (Camb.), F.R.C.S. (Eng.); 1956. London: H. K. Lewis and Co., Limited. 8" x 5¼", pp. 270, with 132 illustrations. Price: £1 15s.

JUST as an experienced fisherman might recount selected piscatorial experiences of his good and bad days, so has Dr. Alex Roche compiled this volume from his case-book. Each story is complete in itself, often with a follow-up of many years and the author's final comments. This "haphazard array of cases" is interspersed with a little philosophy and didacticism.

Any urologist might browse through the pages of such a book with profit and pleasure, as it were mentally discussing personal experiences with the author. Unfortunately the use of abbreviations is excessive; there is no pleasure in reading "M.S.U. (17.12.46) N.A.D." and the like. Moreover the X-ray reproductions are not sufficiently clear for the reader to follow easily the descriptions in the text. Possibly the very obvious honesty of the author prevented him from having the pictures retouched to make them clearer.

Some of the author's methods and opinions may be open to criticism. There is an exceptionally high proportion of intentional or accidental intraperitoneal excursions for a

urologist, and it seems that the transperitoneal route is usual in nephrectomy for any large kidney. For prostatectomy the reader is enjoined to "strictly adhere to certain indications for operation and avoid advocating it in their absence". These are: "repeated attacks of retention; a large or increasing amount of residual urine; nocturnal frequency six or more times; marked hæmaturia; complications. . . ." Just why, in this age, the benefits of prostatectomy should be denied except to the hardy souls who can survive such rigours is beyond comprehension.

Nevertheless, Dr. Roche has many unusual and some unique cases; there are numerous useful hints and a leavening of humour. They would be more enjoyable if they were presented more readably.

Peripheral Vascular Disease. By A. J. Barnett, M.D., M.R.C.P. (London), M.R.A.C.P., and J. R. E. Fraser, M.D., M.R.A.C.P.; 1955. Melbourne: University Press. 8¼" x 5½", pp. 233, with many illustrations. Price: £2 17s. 6d.

THIS book is a short survey of the present concepts of peripheral vascular disease based on the authors' extensive experience in investigation and treatment. It is freely illustrated by case histories. As the clinical picture, whether it be intermittent claudication, disturbance of the colour, temperature or trophic changes in the skin, or the more dreaded rest pain, is the same, regardless of the cause, and depends on the same physiological disturbances, the authors have approached the subject as a single entity. The various ætiological factors—embolism, spasm, atherosclerosis, Buerger's disease *et cetera*—are introduced appropriately as the subject is developed.

The physiology of peripheral blood flow is dealt with fully and is used subsequently as a basis for discussion of symptoms. Diagnostic procedures to determine efficiency of the peripheral vessels, both routine measures and research techniques, are described in detail. The section on treatment is short, reflecting the limitations of present knowledge. The results of treatment and prognosis are supported by the authors' own results. There is a full bibliography.

This book will serve as a helpful guide for the diagnosis and everyday management of patients suffering from this group of diseases, as well as an introduction for those interested in more detailed study of the subject.

The Year Book of the Eye, Ear, Nose and Throat (1955-1956 Year Book Series). Edited by Derrick Vail, B.A., M.D., D. Oph. (Oxon.), F.A.C.S., F.R.C.S. (Hon.), and John R. Lindsay, M.D.; 1956. Chicago: The Year Book Publishers, Incorporated. 5" x 7½", pp. 471, with many illustrations. Price: $6.50.

THIS Year Book maintains the standard of usefulness that practitioners have learned to expect from this series. In it are abstracted articles from relevant journals received by the editors between October, 1954, and September, 1955. The section on the eye has, as previously, been edited by Derrick Vail. It covers the same headings as before, and is actually two pages shorter than in the previous edition. The admonitions and suggestions of the editor are, as usual, very much to the point.

In the section on the ear, nose and throat, the editor, John R. Lindsay, has made some alterations. In the subsection on the ear, the heading "inflammatory ear disease" has replaced "*otitis media*", and stapes mobilization has been added to the subheading "otosclerosis and fenestration". The subheading "facial paralysis" has been dropped, but consideration of the salivary glands has been transferred to the section on the nose and throat. This last section has been expanded somewhat. Additions to the subject matter include the mouth, the neck and tracheotomy. The œsophagus has been given a subheading of its own.

This Year Book will provide ready reference to most modern work on subjects within the specialties with which it has to do. It will be found of value by all who deal with the eye, ear, nose and throat, whether specialists in these fields or not.

Tropical Medicine for Nurses. By A. R. D. Adams and B. G. Maegraith; 1955. Oxford: Blackwell Scientific Publications. 9" x 6", pp. 324, with 34 illustrations. Price: 30s.

IN their introduction to "Tropical Medicine for Nurses", A. R. D. Adams and B. G. Maegraith state that "this is not a book on nursing, so it may be held to aggravate what is said to be a current fault in nursing training—an endeavour to make good nurses into inferior doctors". They "accept this charge"—and justly so; yet how easily could this have been a book to make good nurses into better nurses. These authors go on to state: "The nurse in the absence of a doctor's services must know how to diagnose malaria provisionally, what steps to take to confirm the diagnosis scientifically when facilities later are available, and how to treat the patient with drugs promptly and efficiently to obviate a pending fatality." The implication throughout is that the nurse is not expected to be trained in microscopy; yet in the section on malaria the description of the clinical picture is largely dependent on the microscopic picture, and these statements appear: "If the patient has been taking an antimalarial drug its administration should be stopped until parasites are found in the blood or new treatment is commenced. The clinical picture of regularly recurring febrile paroxysms and an enlarged spleen in an individual recently exposed to infection is very suggestive, but the *certain diagnosis of malaria can be made only by the demonstration of E forms of parasites in the blood.* [The authors' italics.] Identification of gametocytes alone (Frontispiece) is insufficient to establish the diagnosis of active malaria." This is excellent scientific advice for a doctor with all facilities, but how frustrating to the nurse without them! This unhappy state of affairs exists in most sections of the book; more clinical information and less pathological data would make it more truly a book for good nurses, instead of one for inferior doctors.

Conceptions of Modern Psychiatry: The First William Alanson White Memorial Lectures. By Harry Stack Sullivan, M.D., with a foreword by the author and a critical appraisal of the theory by Patrick Mullahy; 1955. London: Tavistock Publications, Limited. 8" x 5½", pp. 311. Price: 32s. 6d.

The Interpersonal Theory of Psychiatry. By Harry Stack Sullivan, M.D., edited by Helen Swick Perry and Mary Ladd Gawel, with an introduction by Mabel Blake Cohen, M.D.; 1955. London: Tavistock Publications, Limited. 8" x 5½", pp. 411. Price: 35s.

IN "Conceptions of Modern Psychiatry", delivered in 1939 as the first William Alanson White Memorial lectures, the late Harry Stack Sullivan expounded his own conception of psychiatry as the study of interpersonal relations, a view which was subsequently modified and expanded in the light of discussions, seminars and clinical investigation. A personality can never be dissociated from the social *milieu* and the field of psychiatry, which, in Dr. Sullivan's view, is not restricted to the mentally sick, but is, to quote his words, "the study of processes that involve or go on between people" and "a fundamental discipline for all those fields that deal with the performance of man". For psychiatrists to set up as authorities and arbiters in the affairs of mankind is a claim which surely invites a challenge. In a somewhat platitudinous review of infancy and adolescence the author pursues his theme of acculturation, preferring this term to maturation. In accordance with current emphasis, Sullivan paid special attention to the relationships between infant and mother (or mother substitute), the "mothering one", with the nipple as "the first of all vividly meaningful symbols". In the interpretation of what goes on in the mind of the infant, Sullivan relied on the memories of his patients, with all fallacies inherent in this form of investigation. As the personality begins to crystallize during adolescence various types emerge, described by Sullivan as the psychopathic self-absorbed, incorrigible, ambition-ridden, chronically adolescent and inadequate, to name a few. The stammerer uses his "vocal misbehaviour" in order to dominate. The use of labels such as schizophrenia and neurasthenia is criticized for the lack of reference to the social relationships which play such a large part in causation and symptoms. Memory is described as the fixation of the pattern of organism-and-environment at some significant time. Treatment must always be directed to "the underlying barrier to full personal intimacy" rather than to more superficial maladjustments, and takes place on psychoanalytical lines by facilitating the accession to awareness of "significant information". New techniques of psychotherapy are constantly needed to meet changing social conditions, but Dr. Sullivan found full doses of alcohol effective in obliterating conflict in schizophrenics during difficult phases of their psychotherapy. He deplored the attempt to escape from feelings of insecurity into trivialities in spite of "remarkable developments in education".

The "interpersonal theory of psychiatry" covers the development of Dr. Sullivan's views on social aspects of psychiatry considered as "an expanding science concerned with the kinds of events or processes in which the psychiatrist participates, while being an observant psychiatrist". He called attention to the modes of communication and the use of language and other symbols between

individuals, and held that anxiety is responsible for inadequate communication and so for a large portion of mental disorders. In spite of being critical of much current psychiatric terminology and aiming at expressing his views in everyday language, he could not avoid the introduction of some new terms to describe his concepts of three main modes of experience, namely: prototaxic, before the use of symbols; parataxic, the use of symbols in a private or autistic fashion; and syntaxic, the use of symbols in the general social sense. Dynamism, dealing with vital activities, is covered in some fifty pages. The theme of acculturation is developed further. According as the infant is satisfied or frustrated at the breast, so is the mothering-one "good" or "bad" and the pattern for later interpersonal relationships is laid down. Dr. Sullivan decided that he could dispense with a good deal of conventional history-taking in order to concentrate on the circumstances of development of anxiety, to be found in the family group and especially in infant-mother relationships. Mental disorders are described in Part III: "Patterns of inadequate or inappropriate interpersonal relations."

Dr. Sullivan was a fluent speaker, and delivered his lectures, mostly from notes, in an easy conversational style. The editors who prepared this second volume worked under difficulties, as Sullivan's views were never static. These two books, so full of "wise saws and modern instances", will be read critically but with interest and benefit by psychiatrists, clinical psychologists and social workers.

Essays in Biochemistry. Edited by Samuel Graff; 1956. New York: John Wiley and Sons, Incorporated. London: Chapman and Hall, Limited. 6″ × 9″, pp. 355, with illustrations. Price: $6.50.

IN this book are twenty-five essays written by twenty-seven of his former students and associates in honour of H. T. Clarke on the occasion of his retirement as Professor and Chairman of the Department of Biochemistry, College of Physicians and Surgeons, Columbia University, New York. The essays cover a wide range of subjects of biochemical interest such as "Some Metabolic Products of Basidiomycetes", "Heterogeneity of Desoxyribonucleic Acid", "The Biochemistry of the Bacterial Viruses", "On the Nature of Cancer", "The Biochemistry of Ferritin", "The Biosynthesis of Porphyrins" and "The Chemical Basis of Heredity Determinants". The essays not mentioned are of equal interest. The writers are all prominent workers in the subjects about which they write. They were given a free hand to review present knowledge or speculate about the future or, as practically all have done, both. Much of the writing is very free so that the reader gets an idea of the personalities of the writers. Thus E. Chargaff, in an essay on "The Very Big and the Very Small", writes of experimental approaches: "So called model experiments often are carried to incredible lengths, prompting one to say that confusion superimposed on complexity may produce papers but not results, and that a skunk dipped in chlorophyll is not yet an apple tree." The papers are all interesting; some are harder to read than others and require, in the reader, a good knowledge of chemistry, but most can be read by anyone with a good general knowledge of biochemistry. For the advanced student in biochemistry and for the research worker the book is a collection of delightful essays in a wide field. Some of the papers could be read with interest and advantage by a thoughtful physician, although there is no "medical" biochemistry in the book. This is a book to be bought and read carefully.

Ciba Foundation Symposium, Jointly with The Physiological Society and The British Pharmacological Society, on Histamine, in Honour of Sir Henry Dale, O.M., G.B.E., M.D., F.R.C.P., F.R.S. Editors for the Ciba Foundation: G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Cecilia M. O'Connor, B.Sc.; 1956. London: J. and A. Churchill, Limited. 9¼″ × 8″, pp. 488, with illustrations. Price: 50s.

THE latest volume published by the Ciba Foundation departs from the previous publications in that it records papers from a symposium of the Physiological and Pharmacological Societies as well as papers and discussions presented at a symposium of the Ciba Foundation. The Ciba Foundation invited the presence, and paid the expenses, of a number of prominent workers from Europe and America. The first symposium was restricted to formal papers, the second to a few papers and much discussion by a small group of investigators of note.

The first symposium was divided into three parts: (i) the occurrence of histamine in the body with four formal papers and five short communications; (ii) release of histamine with five papers and eight short communications, and (iii) the origin and significance of histamine in the body with six papers and seven short communications. The short communications were short accounts of some aspect of work mentioned in formal papers. There is much overlapping here.

All the papers are by prominent workers and cover a great deal of what is currently known about histamine in the body.

The symposium of the Ciba Foundation, in which certain of the speakers in the first symposium gave more advanced accounts of certain aspects of the subject, was divided into two sections: (i) the origin and fate of histamine in the body with six papers and (ii) location of histamine in the body and mechanism of histamine release with four papers. There was a long discussion after each paper and a long general discussion at the end. Probably everything that is known now about histamine is presented in this volume. Histamine was discovered and many of its properties were elucidated by Sir H. Dale in 1910, and this volume has been dedicated to him. He took an active part in the discussions at the Ciba symposium.

While a great deal remains to be done before the purpose and function of histamine in the body are known, this volume gives a splendid account of present knowledge and possible future developments. The physician interested in allergy and anaphylaxis as well as the general physician will find much here to demand his attention.

Psychopathology of Childhood. Edited by Paul H. Hoch, M.D., and Joseph Zubin, Ph.D.; 1955. New York, London: Grune and Stratton. 8½″ × 5½″, pp. 310. Price: $6.00.

THIS is a report of the annual meeting of the American Psychopathological Association, which appears to be a very select body, as there are well under 100 members in the United States of America and Canada, in contrast to the American Psychiatric Association with its thousands of members. What is lacking in quantity, however, seems certainly to be made up in quality. The papers presented are in four groups, each with a critical summary, and are all of a high level. The first section is left for psychologists and deals with problems of development and the difficulties of research, while the other sections are mainly in the hands of psychiatrists such as Levin, Benda, Mahler, Goldfarb, Levy and Kanner.

It is reassuring to find under psychopathology a discussion such as Benda's on the causation of mental deficiency, and there is throughout a recognition of the role of somatic factors in the causation of abnormal behaviour. One contributor (Angus, page 144) states: "The importance of physiological and anatomic considerations in psychopathology lies not only in their theoretic aspects but even more in their practical corollaries. One of the most obvious of these implications is that psychiatry, including child psychiatry, is a branch of medicine, and fundamentally we who are responsible for the care of children with psychiatric disorders are doctors, and the duty and responsibility of the doctor is the study and treatment of the whole patient." Moreover, where the aproach is along psychological lines there is noted a further illustration that American psychiatry is moving away from orthodox Freudian interpretations and replacing these with a much broader based conception of human development and behaviour. As another contributor (Baldwin, page 26) states: "In this recent period our science has been largely living off the hypotheses coming from psychoanalysis. Some of them have been indigestible; others have been digested so thoroughly that they are hardly identifiable; and many form an integral, obvious part of our current beliefs." There is certainly nothing static in the theories put forward, but the dynamics are such as not to place too great a strain on one's credulity, which is not the case with many discussions on the psychopathology of childhood.

It cannot, of course, be claimed that these papers give a complete survey of all aspects of psychopathology, but it can be stated that for those who wish to have an authoritative and clear discussion on many of the problems presenting in this field this book can be thoroughly recommended.

A Handbook of Ophthalmology. By Humphrey Neame, F.R.C.S., and F. A. Williamson-Noble, F.R.C.S. Eighth Edition; 1956. London: J. and A. Churchill, Limited. 6″ × 9¼″, pp. 371, with many illustrations. Price: 30s.

THE appearance of the eighth edition of this book is an indication of its popularity. However, it can hardly be said

to have filled its intention, as written for undergraduates and general practitioners.

The preface makes reference to the fact that the tantalum implant mentioned in the seventh edition is superseded in this edition by the Arruga implant, and that the description of the operation for the intracapsular extraction of the cataract has been rewritten. These matters and many others mentioned in the text are of scant interest to the undergraduate or general practitioner.

Generally the subject matter dealt with is adequately described when one considers the size and intent of the volume, but there is lack of balance. The chapter on operative surgery could be deleted and more space given to ocular manifestations as they touch upon neurology, medicine and surgery. For example, there is no mention of pink disease, most sufferers from which may present to the general practitioner with photophobia. The medical course has grown tremendously in the last ten years and there is a limit to what the student should be asked to learn. We look forward to the day when a text-book intended for undergraduates and general practitioners will deal with the eye problems encountered in general practice, particularly their diagnosis and brief treatment and give an indication as to when the patient should be referred to a consultant, a book which also will contain a description of all the treatments and procedures which a general practitioner may be expected to perform.

The authors perpetuate the mistake of spelling angioma as "angeioma". The statement that pterygium occurs in middle-aged people, to the apparent exclusion of other age groups, is not acceptable, and the omission of the use of cortisone in iritis is unfortunate. The authors have seen fit to describe toxoplasmosis in two separate chapters, but make no mention of intracerebral calcification without which one would hesitate to diagnose the condition. Similarly a discussion on buphthalmos without reference to its treatment by goniotomy, but recommending corneo-scleral trephining, and in addition perpetuating the old theory of its causation, is to be deplored. Finally, we wonder whether anyone recommends the sucking of six lemons a day to patients with thrombosis of the central retinal vein?

We think that these errors and omissions arise from the fact that perhaps the authors have not advanced with the increasing complexities of the undergraduate course. This has changed so much that authors must be careful to adjust their books to meet these altered conditions and should not revise parts of a book the foundations of which were laid some thirty years ago.

In spite of all these criticisms, Neame and Noble probably remains the best available introductory book on ophthalmology.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Annual Epidemiological and Vital Statistics, 1953"; 1956. World Health Organization, Palais des Nations, Genève. 11" x 9", pp. 571. Price: £2 10s.

Here are given 74 tables with details of the most important aspects of the health situation in all parts of the world.

"Virus Diseases and the Cardiovascular System: A Survey", by Ernest Lyon, M.D.; 1956. New York and London: Grune and Stratton. 8½" x 5½", pp. 222. Price: $5.75.

The author has "tried to systematize and integrate past and current writings on cardiovascular involvement associated with viral diseases".

"Techniques in Blood Grouping", by Ivor Dunsford and C. Christopher Bowley, M.B., B.S., M.R.C.O.G., with a preface by R. R. Race, F.R.S.; 1955. Edinburgh and London: Oliver and Boyd. 9" x 5½", pp. 264. Price: 21s.

Intended primarily for the medical laboratory worker.

"Good Health with Diabetes: A Patient's Handbook", by Ian Murray, M.D., F.R.F.P.S.G., F.R.C.P.E.; Third Edition; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 5" x 7", pp. 51. Price: 2s. 6d.

A useful book for diabetic patients to possess, with useful diet tables and advice for occasions of emergency.

"Proceedings of the Round Table on Lysergic Acid Diethylamide and Mescaline in Experimental Psychiatry", held at the annual meeting of the American Psychiatric Association, Atlantic City, New Jersey, May 12, 1955. Edited by Louis Cholden, M.D., Chairman; 1956. New York and London: Grune and Stratton. 8½" x 5½", pp. 95. Price: $3.00.

There are 12 sections and 13 contributors to the discussion.

"Psychosomatic Aspects of Surgery", edited by Alfred J. Cantor, M.D., and Arthur N. Foxe, M.D.; The Proceedings of the First Annual Meeting of the Academy of Psychosomatic Medicine, held in New York City, October, 1954; 1956. New York and London: Grune and Stratton. 8½" x 5½", pp. 232. Price: $6.50.

There are 13 chapters by different authors; these are followed by a summary of the conference.

"Cytology of the Blood and Blood-Forming Organs", by Marcel Bessis; translated by Eric Ponder; 1956. New York and London: Grune and Stratton. 10" x 7", pp. 661, with illustrations. Price: $22.00.

The author's object is to reassess the classical ideas on blood cytology by considering them in the light of recent techniques.

"Bing's Local Diagnosis in Neurological Diseases", by Webb Haymaker, M.D., with chapters by Richard G. Berry, M.D., Bernard S. Epstein, M.D., and Paul I. Yakovlev, M.D.; Second Edition, translated, revised and enlarged from the fourteenth German edition; 1956. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 9½" x 7", pp. 478, with 225 illustrations. Price: £9 4s. 3d.

An extension of Professor Robert Bing's "Kompendium" which appeared in 1909.

"My Story: Memoirs of a New Zealand Nurse", by Mary I. Lambie, C.B.E., with a foreword by Michael H. Watt, C.B.E., M.D., D.P.H., F.R.A.C.P.; 1956. Christchurch: N. M. Peryer, Limited. 9" x 6¼", pp. 100, with illustrations. Price: 30s.

The author was formerly Director of the Division of Nursing in the New Zealand Department of Health.

"Blakiston's New Gould Medical Dictionary", edited by Normand L. Hoerr, M.D., and Arthur Osol, Ph.D., with the cooperation of an editorial board and 88 contributors; Second Edition; 1956. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 9½" x 7", pp. 1489, with 252 illustrations. Price: $11.50.

The book includes "the terms used in all branches of medicine and allied sciences, including medical physics and chemistry, dentistry, pharmacy, nursing, veterinary medicine, zoology and botany, as well as medicolegal terms"

"The Pathology and Surgery of the Veins of the Lower Limb", by Harold Dodd, Ch.M. (Liverpool), F.R.C.S. (England), and Frank B. Cockett, M.S. (London), F.R.C.S. (England), with a foreword by R. R. Linton, M.D.; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 9½" x 7", pp. 471, with illustrations. Price: 65s.

The authors have written with the practical aspects of the subject in mind. "No finer training ground for surgery is to be found than in dealing with venous conditions."

"Histological Appearances of Tumours: With a Consideration of Their Histogenesis and Certain Aspects of Their Clinical Features and Behaviour", by R. Winston Evans, T.D., B.Sc. (Lond.), M.R.C.S. (Eng.), L.R.C.P. (Lond.); 1956. Edinburgh and London: E. and S. Livingstone, Limited. 10" x 7½", pp. 789, with 989 illustrations. Price: 90s.

The book is concerned "not only with the histological and histogenetical aspects of tumours but also with the development of the tissues and organs from which they arise".

"Progress in Radiobiology: Proceedings of the Fourth International Conference on Radiobiology Held in Cambridge on 14th to 17th August, 1955", edited by Joseph S. Mitchell, Barbara E. Holmes and Cyril L. Smith; 1956. Edinburgh and London: Oliver and Boyd. 9½" x 6", pp. 598, with illustrations. Price: 63s.

Divided into eleven sections, in each of which are several subsections and discussions.

"Lectures on the Scientific Basis of Medicine"; British Postgraduate Medical Federation, University of London; Volume IV, 1954-55; 1956. University of London: The Athlone Press. 8½" x 5½", pp. 406, with illustrations. Price: 37s. 6d.

Comprises a selection from the winter lectures of the British Postgraduate Medical Federation.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 15, 1956.

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COMPOSTING.

THE use of wastes, particularly human and animal excreta, has, from very ancient times, been associated with the improvement of soil fertility, and indeed has been a major factor contributing to the survival of some of the ancient civilizations. With this has been associated disease in man, particularly intestinal disease. The older methods, still in use in many countries, were very insanitary, and in areas where night-soil is commonly used as fertilizer, it is not unusual to find more than 90% of the human population infested with one or more of the several intestinal parasites—bacterial, amœbic or helminth.

Community and human wastes are of undoubted value as fertilizers, but safe and sanitary methods must be evolved for their use. Composting, properly controlled, is such a method. The art of composting is very old, but it is only in recent years that intensive scientific studies have made clear the underlying biological and chemical processes involved. Modern methods of composting were first suggested in India by Sir Albert Howard about thirty years ago. The World Health Organization is interested in the maintenance of soil fertility, provided the methods used are consistent with acceptable standards of public health. WHO commissioned H. B. Gotaas, Professor of Sanitary Engineering at the University of California, a man who has done considerable research on composting and has advised on the erection and running of plants in many parts of the world, to prepare a comprehensive report on composting, particularly from the sanitary disposal point of view. The result is a book of 205 pages called "Composting: Sanitary Disposal and Reclamation of Organic Wastes".[1] While the stress is largely on the sanitary aspect, other aspects are treated adequately and the result is a work that can be read with profit and interest by any intelligent person. Much of what has been written on composting consists largely of magic, mystery and mumbo-jumbo, but this book is thoroughly scientific.

Composting of organic waste materials can be carried out aerobically, anaerobically or by a mixture of the two. In these processes bacteria, fungi, moulds and other saprophytic organisms use the organic material for food and convert it into more stable forms. The waste material used may be animal and human excreta, vegetable matter such as weeds, clippings *et cetera*, household refuse, organic industrial wastes or any mixture of these. In the aerobic process organisms, using oxygen, feed upon the organic matter, using nitrogen, phosphorus, and some of the carbon and other elements to make cell protoplasm. About two-thirds of the carbon in the original material passes off as carbon dioxide; the rest is largely combined with nitrogen. When ample oxygen is available there are no smells. A great deal of energy is set free in the form of heat during the conversion of combined carbon to carbon dioxide. The interior of a pile of decomposing material may rise to over 70° C. When temperatures over 45° C. are reached thermophilic bacteria take over. The temperatures reached under these conditions rapidly kill pathogenic organisms, worm eggs and weed seeds. Aerobic decomposition or composting can be carried out in silo digesters, pits, bins, stacks or piles, or in special apparatus if adequate oxygen is supplied. This can be obtained by frequent turning. In anaerobic composting putrefactive breakdown of organic material takes place. The waste carbon here is mainly converted to methane with some carbon dioxide. This is the sort of fermentation which takes place in a marsh. There are always objectionable odours produced by such substances as hydrogen sulphide and mercaptans. Very little heat is engendered in this process—a disadvantage for the killing of pathogens. Given time, these are destroyed, but only after some months. Weed seeds and helminth eggs are not all destroyed. In the mixed aerobic-anaerobic process the material, usually in a pile or pit, is turned at infrequent intervals. This is the commonest process, particularly in composting on a small scale, and is effective, but takes longer than the aerobic method. In an aerated digester or when piles are turned frequently the composting process is complete in three days to three weeks, when the piles are turned infrequently in up to six weeks, and when the piles are not turned in from four to six months. Ascaris eggs seem to be the most difficult to kill, but three turns of a pile in thirty-six days killed all ascaris eggs.

The case for the use of composting as a means of destruction of pathogenic organisms with consequent great improvement in health, particularly in smaller communities, seems to have been well made out. The other aspect of composting is the improved nutrition which can be obtained when the wastes, in more acceptable form, are returned to agricultural land to provide plant nutrients. Waste organic materials come in the long run from vegetation, and contain nitrogen, phosphorus, potassium and

[1] "Composting: Sanitary Disposal and Reclamation of Organic Wastes", by Harold B. Gotaas; 1956. Monograph Series, Number 31. World Health Organization, Palais de Nations, Geneva. Price: £1 5s.

trace elements—all necessary for continued fertility of the soil. Compost, properly prepared, puts these elements back into the soil. The physical properties of humus and the organic material left in the ripened compost are of great importance in the soil. Soil fertility depends on soil structure as well as on nutrients. The humus increases the degree of aggregation of soil particles and so increases the water retention capacity of the soil, and also encourages the more extensive development of the root system of plants. The nitrogen present in the compost is largely in bacterial protoplasm, and as the bacteria die in the soil the nitrogen is gradually set free for the plants. The aggregation of the soil particles is brought about by cellulose esters (cellulose acetate, methyl cellulose and ethyl cellulose). These factors are sufficient to explain the value of compost in improving the fertility of soil, and there is no need to look for mystical or magic properties, hormones and the like. The author shows that the addition of much-advertised inocula or activators to compost in the making serves no useful purpose, and millions of tons of compost are made successfully all over the world without any additions.

A considerable part of the book is taken up by descriptions of plants used by various public bodies throughout the world for composting various types of organic wastes. Details are also given of small-scale methods for individuals or small communities. The author points out that in assessing the cost of composting the public body must consider that it has to get rid of the waste material somehow, and the cost of incineration or other methods should be considered. Many hundreds of municipalities or other public bodies all over the world are disposing of their waste material at a profit by composting. The largest scheme of the kind in New South Wales seems to be at Canterbury, near Sydney, and is apparently very successful.

The author has made a very good case for the utilization of waste organic materials for composting as a means of reducing human infections and for the improvement of fertility of the soil for agriculture.

Current Comment.

RAUWOLFIA IN HYPERTENSION.

EXTRACTS of the plant *Rauwolfia serpentina* had been used for the production of hypotension in Indian medical practice for ten years before serious attempt was made to subject the various resulting alkaloids to clinical comparison. In 1949 R. J. Vakil[1] used extracts of the dried root in the treatment of benign essential hypertension. Since this time various derivations of the dried root have emerged into clinical use and have largely replaced the crude extract, though a comparison of the clinical effectiveness of combinations of alkaloids and crude extract by H. Tuchman and C. W. Crumpton[2] revealed no significant differences between them.

W. R. Livesay[3] found *Rauwolfia serpentina* to be useful in all cases of essential hypertension, but in only the most mild types was it sufficient alone. The sole use of rauwolfia in the mild cases of essential hypertension was also advocated by E. D. Freis.[4]

[1] *Brit. Heart J.*, 11:350, 1949.
[2] *Am. Heart J.*, May, 1955.
[3] *J.A.M.A.*, July 17, 1954.
[4] *M. Clin. North America*, March, 1954.

The therapeutic effect of reserpine taken by mouth is slow to develop. However, giving reserpine by the intravenous or intramuscular routes, W. M. Hughes *et alii*[5] were able to produce a rapid lowering of blood pressure levels in patients severely ill with such conditions as malignant hypertension, severe benign hypertension, cardio-vascular disease, toxæmia of pregnancy, and acute glomerulonephritis. The psychiatric side effects were noted by F. H. Smirk and E. G. McQueen[3] and by R. W. Griffin *et alii*.[3] Depression is a particularly troublesome sequela to the use of reserpine, and nightmares, insomnia and suicidal tendencies are not uncommon.

Some authors, such as R. W. Wilkins,[4] tend to regard the good effects of the rauwolfia derivatives as part of the sedative effect of these drugs, and in view of their effect on the mental diseases of tension, and of the effect of mental tension on the blood pressure, this view is not surprising. The view that reserpine exerted its effect upon cortico-visceral function is supplemented by the views of A. J. Mjasnikov,[5] who suggests, on an extension of the work of Pavlov, that essential hypertension is the expression of a peculiar neurosis directed towards the vascular system and originating in the stresses of earlier life. However, it is noted that G. W. Pickering[6] presents interesting statistical evidence on the hereditary nature of essential hypertension. In fact, of course, these two views are not so irreconcilable as they may at first appear. The sedative effect of reserpine has not been found to be of use in the treatment of menopausal symptoms by H. S. Kupperman *et alii*.[6] Moreover, as E. G. McQueen and G. L. Blackman[7] have shown, reserpine and the related alkaloids have a direct vasodilatory effect upon isolated blood vessels, and differences in the speed of activity appear to be related to the different solubilities of these compounds.

The use of rauwolfia derivatives was discussed in some detail at the Third International Congress of Internal Medicine held in Stockholm. F. C. Lorenzo[8] suggests that *Rauwolfia serpentina*, as well as being useful in patients with mild hypertension, is also valuable in the preparation and stabilization of patients with severe hypertension prior to the administration of more potent hypotensive agents. Lorenzo suggests that the site of action of reserpine is in the hypothalamus. A. H. Douthwaite[3] has found particular benefit in patients with severe benign hypertension by the combination with reserpine of hydrazinophthalazine or of pentapyrrolidinium. E. Donzelot[3] uses reserpine in combination with the hydralazines; small doses of each can be used, as it would appear that, in combination, there is potentialization of the effect. R. Meier, H. J. Bein, F. Gross and J. Tripod[3] suggest that "Serpasil", regardless of the point of attack, abolishes the hypotensive response to centripetal stimulation, but, unlike other drugs in use, does not inhibit the hypertensive effect of central stimulation. However, there is insufficient information available to confirm the specific usefulness of the different drugs in the different types of hypertension. C. H. Kelchner[3] has found rauwolfia to be effective only in mild cases of essential hypertension. Of patients with more severe disease, 75% are treated satisfactorily by the combination with *Rauwolfia serpentina*, of phenoxybenzamine hydrochloride, which is a peripheral adrenergic blocking agent, and of protoveratrine, which acts on the vasomotor centre and vagus nerves to produce general vasodilatation and bradycardia. Therapeutically, the effect is equal to that of hexamethonium and other like compounds with severe side effects, though C. Bartorell[3] has found that in some patients "Serpasil" is more effective as a hypotensive agent than hexamethonium itself.

[1] *Arch. Int. Med.*, April, 1955.
[2] *Lancet*, July 16, 1955.
[3] *New England J. Med.*, March 29, 1956.
[4] *J. Chronic Dis.*, May, 1955.
[5] *Acta med. scandinav.*, Supplement 312, 1956.
[6] *J. Am. Geriat. Soc.*, April, 1956.
[7] *Australasian Ann. Med.*, November, 1955.
[8] *Acta med. scandinav.*, Supplement 312, 1956.

H. L. Drezner and S. Horoschak[1] have reviewed their clinical experience in the treatment of hypertensive patients with the alseroxylon fraction of *Rauwolfia serpentina*. The tranquillizing effect of the drug produced rapid control of the associated symptoms. Compared with reserpine, the side effects were slight. The alseroxylon derivative produced moderate hypotensive effects in all types of essential hypertension and in patients with fixed malignant hypertension. T. M. Feinblatt *et alii*[2] have found that the distressing side effects of rauwolfia preparations were effectively relieved by the concurrent administration of small doses of ephedrine. They suggest that ephedrine, in this treatment, does not result in the cancellation of the hypotensive effects of rauwolfia.

R. S. Green and D. Davolos[3] have made a long-term study of the effect of crude *Rauwolfia serpentina* and of its alseroxylon fraction in the treatment of patients with hypertension. They suggest that the drug should be administered at bedtime, and a satisfactory response may occur at any time within two weeks of treatment. Sedation, nasal congestion and nightmares, which may occur, can usually be controlled by a reduction of dosage. No constant changes were noted in the electrocardiographic findings, though radiological studies frequently revealed some decrease in cardiac hypertrophy, and there was occasional reduction in the transverse diameter of the enlarged aorta. No disturbance of renal or liver function occurred. No correlation between types of patients and their physical signs and the response to the drug was found. For patients in whom the clinical response was not entirely satisfactory, the subjective symptoms were usually considerably relieved. Unusual complications of therapy were peripheral neuritis and amenorrhœa.

R. Platt and H. T. N. Sears[4] suggest that in the absence of the ideal drug for the treatment of hypertension, patients with the milder conditions should not be treated by hypotensive drugs. Of 41 newly treated hypertensive patients, the response was good in 17, and there was no effect in 16. Of 13 cases in which "Serpasil" was added to previous therapy by ganglion-blocking drugs, the result was good in nine. Vision improved in cases of retinopathy. No postural effect on the blood pressure was found, and the tranquillizing effect and the slowing of the pulse were general. Significant improvement followed in an average of four weeks after the commencement of treatment. Drowsiness and nasal congestion occurred in half the patients and diarrhœa occurred in four cases. In three female patients lactation occurred which ceased spontaneously in two. The most dangerous side effect was depression, and for this reason the treatment was stopped in 10 patients. Most of these had previous histories of mental instability, and one patient committed suicide.

R. L. Hershberger, E. W. Dennis and J. H. Moyer[5] have investigated the effect of one of the alkaloid extracts of rauwolfia, which has been called rescinnamine. The actions of this drug resemble reserpine, except that rescinnamine is slower in inducing hypotension and the action is more prolonged. Rescinnamine had less slowing action on the heart and there was less gastro-intestinal effect than with reserpine. Apparently a useful hypotensive effect was obtained in about half the patients treated, a finding common to most of the different series of hypertensive patients in which rauwolfia drugs have been used.

The findings of the different workers agree quite remarkably. The rauwolfia drugs have a significant hypotensive effect in a large proportion of hypertensive patients. However, the drugs are dangerous and need rigorous clinical control. Of all the side effects, mental depression is the most important, and a useful lesson is gained by remembering that, even under the care of Platt and Sears, one such patient committed suicide. G. Lemieux, A. Davignon and J. Genest[6] have found that patients who receive rauwolfia therapy for hypertension, 10% develop a depressive state, and some of these require institutional care. However, as R. H. Burrell[1] has suggested, while the mild depressive states may be induced by the reserpine-like drugs, the more severe psychotic depressions are largely of endogenous or constitutional type. Burrell reports that sodium succinate by mouth is effective in combating much of the depression caused or accentuated by reserpine, without affecting the blood pressure. G. J. Sawyer-Foner and W. Ogle[2] suggest that, in fact, the depressive effect of the rauwolfia drugs is unspecific; chlorpromazine has the same effect. The depressive reaction is not part of a selective action in the brain stem, but is the individual psychological response to the removal of one of the patients' important defences, such as increased activity. Whatever may be the final role of the rauwolfia alkaloids in clinical practice, their dual role and their numerous effects form a communication between pure medicine and psychiatry. By such means, a better understanding of psychosomatic factors may lead to the more efficient treatment, and even to the prevention of essential hypertension.

[1] *Internat. Rec. Med.*, May, 1956.
[2] *J.A.M.A.*, June 2, 1956.
[3] *Am. J. Med.*, May, 1956.
[4] *Lancet*, April 14, 1956.
[5] *Am. J. M. Sc.*, May, 1956.
[6] *Canad. M. A. J.*, April 1, 1956.

POSTURE AND THE LUMBAR REGION OF THE SPINE.

In an age which has produced the prolapsed intervertebral disk, spondylolisthesis, ankylosing spondylitis and intervertebral osteoarthritis, the importance of the lumbar part of the spine and of the signs which indicate disease in this area has increased in clinical practice. The lumbar region of the spine tells radiological tales of secondary carcinoma, it is probed for cerebro-spinal fluid, it is the way of entry of the spinal anæsthetic, and it is the seat of postural pain and the repository of pain of functional origin. The lumbar spinal region which was always important to the individual has now become important to medicine, and therefore the normal motions of the lumbar vertebræ have thus gained some deserved attention.

S. S. Tanz[1] has made a radiological study of the lumbar region of the spine in children and adults. He found that there are wide variations in the mobility of the normal lumbar vertebræ. Lateral mobility range is about two-thirds that of the to-and-fro movement, and some subjects have a definite rotation range for the whole lumbar region. Young children have highly mobile lumbar vertebræ, and the study revealed that the decrease of mobility with age is most rapid between the ages of thirteen and thirty-five years. The fifth lumbar interspace is the site of more to-and-fro movement than any other in children; the same is true of the fourth space in adults. The fifth space is the least affected by lateral bending. Absence of lumbo-sacral mobility was found not uncommonly by Tanz, though the lumbo-sacral joint had no tendency to fuse with advancing age.

J. J. Keegan[4] has studied the alterations of the lumbar curve in relation to posture and seating. He states that over the age of thirty years there is an increasing tendency to experience lower lumbar pain associated with postural strain. The physiologically normal position of the adult spine is with the trunk-thigh angle and the knee angle at about 135° each. In this position the lumbar curve is considerable, and there is balanced muscle relaxation with stabilization of the intervertebral disks. Alteration of this curve places a great wedging strain upon the lower lumbar disks. A correct chair should provide a free space below the lumbo-sacral juncture to accommodate the posteriorly projecting sacrum and buttocks. The surface of the lower lumbar support should be convex and the back support of a straight chair should end below the scapulæ. The seat of a chair should be short and the weight should be on the buttocks, not on the thighs, and the feet should be able to be placed beneath the seat for relaxation and as an aid in rising; a high-backed chair

[1] *New Zealand M. J.*, June, 1956.
[2] *Canad. M. A. J.*, April 1, 1956.
[3] *Am. J. Roentgenol.*, March, 1953. Quoted by "Year Book of Orthopædic and Traumatic Surgery, 1953-54".
[4] *J. Bone & Joint Surg.*, July, 1953.

should not cause uncomfortable flattening of the lumbar spinal region. Prolonged malposition of the lumbar spinal region encourages and accentuates the pressure symptoms of a diseased intervertebral disk.

K. L. Ahlman, E. O. Eränkö and P. Virtama[1] have studied the alterations of the lumbar curve and intervertebral spaces related to lying. The 10 subjects used in the study were all healthy, and radiological studies of the lumbar part of the spine were made while each subject was standing erect, and while lying relaxed in different kinds of bed. Three kinds of bed were used—the wire net variety fixed at all sides to an iron tube frame and acting like a hammock, a kapok mattress on a hard board bottom, and a "Latex" foam mattress on the same bottom. The mean distance between the vertebræ differed little from the normal standing position when the subject lay on the hard kapok mattress. The straight "Latex" foam mattress caused a significant narrowing in the intervertebral spaces. A highly significant decrease in the mean vertebral distance, and a highly significant increase in the mean dorsal distance, were caused when the subject lay in the hammock type of bed, and there was a significant decrease in the mean volume of the intervertebral spaces. The lumbar curve was significantly straightened even on the kapok mattress, more so on the "Latex" foam mattress, and by far the most on the hammock bed. The greatest change in curvature occurred at the last lumbar intervertebral space. Ahlman *et alii* consider that the straightening of the lumbar curve and the decrease in volume of the intervertebral spaces while the subject was lying on the hammock type of bed were sufficient to deserve serious consideration. Even a small narrowing of the intervertebral space can be expected to cause a considerable increase of pressure inside the disk. Processes which lead to disease of the intervertebral disks are likely to be facilitated by the wrong kind of bed.

The results of this study confirm the general impression that the sagging bed is an unhealthy bed. The comfort of the soft, all-supporting mattress is also misleading. It is no uncommon experience that once the hard, flat bed has become familiar, despite the apparent lack of comfort, sleep is less disturbed and the familiar stiffness in the back on waking is less noticeable. The hard flat bed is a healthy bed and its regular usage is merely the continuation of a good habit.

[1] *Ann. Chir. et Gynæc. Fenn.*, 44: 4, 1956.

PROGNOSIS IN EMPHYSEMA.

WHILE emphysema is a well-recognized pathological entity, and while its effects are easily recognized during lifetime, its diagnosis in the living subject is not made without some difficulty. Clinical examination of the chest can give no precise information on the extent of the emphysematous changes, and radiological findings are scarcely more helpful. Difficulties thus arise when the clinician attempts to assess the degree of pulmonary lesions, to advise on the wise limitations of activity, and to make a prognosis. Methods of estimating respiratory efficiency are inaccurate, and it proves almost impossible to lay down precise criteria for the recognition of the signs of emphysema at the various stages of disability. R. Marshall, R. W. Stone and R. V. Christie,[2] who investigated the relationship between dyspnœa and the work of breathing, found that the only sensation capable of accurate assessment was the point at which further exercise would produce respiratory discomfort, and which was presumably the limit of respiratory performance. In emphysematous subjects, the force exerted on the lungs might be as great as in the normal subject, but the minute volume respired was greatly reduced and there was increased resistance to respiration owing to increased viscous resistance.

The treatment of pulmonary emphysema has proved to be discouraging. The effectiveness of breathing exercises has been variously reported, and clinical experiences tend to confirm the view that the value of exercises lies in their functional impact on the patient in his approach to dyspnœa and on the evacuation of bronchial secretions. W. F. Miller[1] found that diaphragmatic breathing exercises did decrease the functional residual capacity of the lungs, increased the alveolar ventilation, and improved the velocity of air flow. In his patients there was objective evidence of increased exercise tolerance. E. J. M. Campbell and J. Friend,[2] who studied the electromyelographic changes in the accessory respiratory muscles, concluded that the only benefit which appeared after breathing exercises was that the relief of dyspnœa was more rapid, and that the patient breathed by the exercise method rather than in the natural way.

In an effort to relate respiratory function in emphysematous patients to the prognosis of the disease, D. V. Bates, J. M. S. Knott and R. V. Christie[3] have followed the progress of a series of such patients over a period of several years. Individuals were carefully selected and fulfilled defined criteria. Each had a history of non-seasonal increase in dyspnœa on exertion; there was no cause for the dyspnœa other than emphysema, there was no hypertension and no *cor pulmonale* when the patients were first examined. In addition, each patient was a good witness and there was no functional overlay. There was usually a history of chronic bronchitis. In all, 59 patients were fully investigated over a long period of time. As with several other recent carefully controlled clinical trials, the assessment of physical signs was found to be subject to considerable inaccuracy. There was a fair agreement between observers on the presence or absence of rhonchi. There was a general absence of polycythæmia. The authors concluded that the history was the most reliable guide to the diagnosis. Most of the patients were found to have led sedentary lives, and there were few whose occupation had involved hard physical labour. During the average of the three years' observation of the study there were 17 deaths, whereas from tables of normal life expectation only four could be expected. Of the deaths, 13 were due to respiratory causes, and most of the subjects had given evidence of right heart failure. In surviving patients, dyspnœa was found to be worse in winter months, and particular difficulty with a possible fatal outcome followed fog. The presence of rhonchi was little affected by the season and dyspnœa was not related to the presence of rhonchi. Of the surviving 42 patients, all but 11 deteriorated during the period of investigation. Four methods of assessing respiratory function were used. The first involved measurement of the subdivisions of lung volume and mixing efficiency, but the calculations appeared to have little value and were sensitively altered by variations in the degree of bronchospasm. Estimations of the maximal ventilatory volume were found to be of slight value. Measurements of the diffusing capacity of the lungs, or of the ability of the lungs to aerate the blood, were carried out, carbon monoxide being used with the subjects at rest. The fourth test was that of oxygen saturation of the blood; this proved to be too insensitive for use.

When the results of the tests of respiratory function are compared with the clinical assessment of the patients and the history of deterioration or death, it becomes apparent that only the measurement of the diffusing capacity bears any accurate relationship to the progress of the disease. Other tests could be correlated to the progress of individual patients, but did not closely follow a general pattern of change. Study of the patients in exercise brought an even greater discrimination to the value of the estimations of diffusing capacity. The normal patient may almost double his diffusing capacity with exercise, while the emphysematous patient cannot do so. The functional defect in emphysema is one of gross impairment of the efficiency of ventilation and of overall diffusion within the lung. Increasing severity and hence increasing disability are related to the progressive impairment of gaseous diffusion. It seems likely that the measurement of diffusing capacity is really a direct indication of the degree of destruction of

[2] *Clin. Sc.*, November, 1954.

[1] *Am. J. Med.*, October, 1954.
[2] *Lancet*, February 13, 1955.
[3] *Quart. J. Med.*, January, 1956

the lung parenchyma. Provided that the adverse effects of bronchospasm can be ruled out, measurement of diffusing capacity is a reliable indication of the particular state of respiratory inefficiency reached. It also appeared, from this study, that the onset of right heart failure was heralded by a fall in the diffusing capacity. The authors conclude that while control of bronchial obstruction and bronchospasm may delay further emphysematous changes, it is the reduction in the pulmonary capillary bed which is the real disabling lesion related directly to the prognosis, and satisfactorily assessed by estimations of the pulmonary diffusing capacity.

SURGICAL ALLEVIATION OF PARKINSONISM.

SEVERAL neurosurgical procedures have been reported to improve the condition of patients suffering from post-encephalitic Parkinsonism. The earlier surgical techniques often alleviated the tremor by inducing paralysis in the affected muscle groups. In an attempt to maintain voluntary movement, the newer techniques have become still more difficult, but the results have been encouraging, and while the operations cannot in any way cure the disease they are very well worth while, for they make tolerable, and even useful, the lives of those condemned to one of the most incapacitating and demoralizing of neurological abnormalities. The most satisfactory results have been obtained in patients in whom the Parkinsonism is predominantly unilateral. O. Sjöqvist,[1] in patients in whom the chief manifestation of the disease was tremor, sectioned the lateral pyramidal tract and the antero-lateral column at the third cervical segment of the spinal cord. Motor function was almost completely restored in four to six weeks, and in successful cases there was no tremor, though the rigidity remained. I. S. Cooper[2] reported on the results of occlusion of the anterior choroidal artery in 34 patients. Hemiplegia developed in only two cases. Resting tremor was abolished in most of the patients and cogwheel rigidity was much reduced. Cooper reported that the operative mortality was 10% and that the surgical risk was increased in patients over the age of fifty-five years. The operation was bilateral in six cases.

P. Bailey and O. Sugar[3] quote G. Guiot and S. Brion,[4] who operated on 81 patients by electrocoagulation of the inner pallidum. The boundary between danger and success is narrow, and the results in Parkinsonism were variable. The techniques could not be applied to both sides. More success was reported by F. Fénelon[5] by electrocoagulation of the *ansa lenticularis*.

I. S. Cooper, N. Poioukhine and A. Morello[6] have summarized the two neurosurgical techniques used in advanced Parkinsonism, by surgical occlusion of the anterior choroidal artery and by chemopallidectomy. Each operation has been performed on a series of 50 patients. The authors found that for the operation of occlusion of the anterior choroidal artery, local anæsthesia was preferable to avoid the post-operative complications of somnolence, morbidity and pneumonia. The anterior choroidal artery is coagulated about one millimetre from its origin, and again 1·5 millimetres distally. Clipping alone is not sufficient to ensure complete and permanent obliteration of the vessel, which is the principal blood source of the *globus pallidus* and of the related extrapyramidal nuclei. Of the 50 patients, five died, and good or excellent results were obtained in 30. In several cases there was immediate intellectual or neuro-muscular difficulty, but most of such complications proved to be temporary. Of the 12 patients in whom the operation failed to alleviate the condition, the cause was failure to obliterate the anterior choroidal artery. Particular technical difficulty results from the variability of origin and distribution of the artery despite the pre-operative help of visualization by cerebral arteriography.

The operation of chemopallidectomy, which has been performed by Cooper *et alii* in 50 cases, was evolved from the investigations of the injection of procaine into the *globus pallidus* for the purpose of inducing temporary relief of the contralateral tremor and rigidity. The correct route of operation is determined by preliminary pneumoencephalography, and the actual operation consists of the passing of a polythene cannula at an angle aimed at the top of the third ventricle until its tip lies in the region of the mesial *globus pallidus*. Confirmation of the position of the cannula is obtained by the injection of procaine, and if the expected contralateral relief of tremor and rigidity occurs, 0·4 millilitre of dehydrated alcohol is slowly injected over a twenty to thirty minute period. The cannula is retained in place and the alcohol injection is repeated twice during the ensuing week. Serious consequences of the procedure have been death in two cases, hemiplegia in one instance, and ataxia and oculomotor nerve palsy in another. The overall incidence of good, lasting results has been a little higher than that for the operation on the anterior choroidal artery. By means of both surgical procedures patients severely or totally incapacitated for many years have been returned to a high level of activity.

[1] *Zentralbl. Neurochir.*, 14: 16, 1954; quoted by Bailey and Sugar, *loco citato*.

[2] *J. Am. Geriat. Soc.*, November, 1954.

[3] "Year Book of Neurology, Psychiatry & Neurosurgery, 1955-1956", Year Book Publishers, Chicago.

[4] *Semaine hôp. Paris*, May 26, 1955.

[5] *Ibidem*.

[6] *J.A.M.A.*, April 28, 1956.

DIGESTIBILITY OF LEAN MEAT.

TEACHERS of human nutrition often point out that the cheaper, and usually tough, cuts of mutton and beef are as nutritious as the choicer and dearer cuts provided they are properly prepared for the table. Seldom, however, is information given as to the best methods of preparing the cheaper meats.

J. H. Farrel[1] has studied the effects of various procedures on the digestibility of lean meat. He found the meats may leave up to 85% of their original weight as undigested residues if they are not well chewed. The cheaper cuts of meat are tough and difficult to chew. The methods studied for making the meat more tender and chewable were: (i) the meat was soaked for twelve hours in diluted vinegar—about 4% to 6% acetic acid; (ii) the meat was treated with one of the commercial meat tenderizers containing papain; (iii) the meat was beaten with a rolling pin or spiked hammer; (iv) the meat was beaten with a rolling pin or spiked hammer and treated with a tenderizer. In each experiment the meat—lean steak of the cheaper kind—was cut into two pieces as nearly as possible equal in size and fried, and then each piece was cut into cubes measuring about 1·0 centimetre on each side. Each set of cubes of meat was then sewn into a cotton muslin bag with holes 1·5 millimetres square. The bags were swallowed in pairs and later collected from the fæces excreted in about twenty-four hours. "Normacol" was given to ensure quick passage of the bags so as to prevent too much bacterial decomposition of the residues in the colon. The well-washed residues were collected and weighed. Soaking the meat in vinegar and beating with a rolling pin had no significant effect on the digestibility of the meat. The spiked hammer was very effective, increasing the digestibility on an average by 28% ($P > 0{\cdot}001$), and the tenderizer showed a corresponding figure of 16% ($P > 0{\cdot}01$). When the spiked hammer and the tenderizer were used the digestibility was increased by 37% ($P > 0{\cdot}001$). In another experiment thorough chewing of the meat increased its digestibility by an average of 32%.

While this is difficult to measure, the author's definite impression was that the meat which was made more digestible was tenderer.

[1] *Brit. J. Nutrition*, 10: 2, 1956.

Abstracts from Medical Literature.

PATHOLOGY.

Hyalinization of the Islets of Langerhans.

E. MOSCHCOWITZ (*Arch. Path.*, February, 1956) has studied the lesions of the islets of Langerhans which he states are found in almost 50% of cases of *diabetes mellitus*. Interest has arisen particularly from a study of the much discussed problem of the relationship of *diabetes mellitus* with arteriosclerosis. Gross arteriosclerosis of the pulmonary artery, which is entirely independent of that in the arteries of the greater circulation, occurs no more frequently in diabetics than in control patients. On the other hand, the incidence of atherosclerosis is higher in diabetics than in non-diabetics and is the result of the hyperglycæmia and more particularly the hypercholesterolæmia. Atherosclerosis and arteriosclerosis are different lesions in respect to both morphology and pathology, and they should not be viewed as synonymous. Arteriosclerosis may exist without atherosclerosis. Hypertension itself, whether associated with diabetes or not, increases the incidence of arterial disease in the pancreas. It might appear that diabetics are subject to hypertension and, in consequence, to arteriosclerosis. However, many patients with prolonged hypertension ultimately develop hyperglycæmia and eventually a typical glucose tolerance curve of *diabetes mellitus*. Indeed, it has been held that every patient with essential hypertension is a potential diabetic. There is no evidence that adult diabetics are more subject to hypertension than others. The two lesions that affect the islets of Langerhans are fibrosis and hyaline degeneration. All islets contain fibrous tissue as part of the vascular content and the amount of connective tissue increases with age. With associated hypertension, the thickening of the capillary wall is intensified. The islet lesions in adult *diabetes mellitus* are nearly always associated with arteriosclerosis of the pancreatic vessels and may be interpreted as a capillary sclerosis, precisely comparable to that found in the alveolar capillaries of the lung, in the sinusoids of the liver and in the glomeruli in arteriosclerosis or phlebosclerosis affecting the main supply vessels of these organs. The capillary sclerosis is the result either of the extension of the fibrosing or hyalinizing lesions from the efferent arteriole of the islet into the capillaries, or of the diminution of the blood supply. The lesions do not occur more frequently because of the special free anastomosing pattern of both the grosser and the capillary blood supply of the pancreas and of the islets. The cause of the hypoinsulinæmia in the cases in which the islets are intact is considered by the author to be an insufficiency, consequent to an impairment to the grosser blood supply. No single instance of the islet lesion was discovered in the five cases of juvenile diabetes examined pathologically. This observation and the different clinical behaviour suggest that juvenile diabetes has a different origin from the adult type.

Fatal Hepatitis and Cirrhosis in Infancy

R. PEACE (*Arch. Path.*, February, 1956) has made a comparative study of a series of cases of fatal liver damage in infants occurring both in the years before and in the years after the introduction of the antibiotics. Thirty-two cases were found to be suitable for the purposes of this study. The average age at the time of death from all forms of liver damage was two and a half months. Without respect to the ætiology, it was found possible to form composite clinical pictures of both acute and chronic forms of severe liver disease in infancy. According to the ætiology of the hepatic damage the cases were classified into five groups, and in each group the clinical and morphological characteristics were critically analysed. It was found that cholestatic liver damage and biliary cirrhosis secondary to biliary atresia were commonly diagnosed too often. The several forms of viral hepatitis and post-hepatic cirrhosis often were incorrectly interpreted, even after necropsy, as obscure variants of biliary atresia. Examples of cholangiolitic hepatitis and cirrhosis had not been recognized as such. Interstitial hepatitis and post-necrotic collapse secondary to omphalitis and portal pyæmia were encountered in material from recent years. There was not one case of post-erythroblastotic liver damage that could be substantiated by anatomical lesions. Various forms of giant liver cells were found in association with syphilitic hepatitis and cirrhosis, the viral hepatitides and their sequelæ, cholangiolitic hepatitis and biliary cirrhosis, due to malformation of extrahepatic bile ducts. It was possible to associate giant liver cells with repeated episodes of injury and subacute clinical behaviour patterns.

Antibiotics and Pulmonary Cysts in Tuberculosis.

J. DENST AND W. F. RUSSELL (*Am. J. Clin. Path.*, April, 1956) have studied tuberculous cavities found in the resected specimens obtained from 57 patients. They found that sterilization of open tuberculous cavities was obtained in nine instances by the use of "Isoniazid" and streptomycin. The sputum was rendered clear of tubercle bacilli when the two drugs were used simultaneously and prior to the development of drug resistance by the bacteria. Eight of the lesions were converted into smooth lined cysts; in four of these there was no residual histological evidence of tuberculous activity. The formation of these thin walled cysts seems to depend on the intensive treatment of an exudative lesion, before there is much fibrosis, and before before drug-resistance develops. The cysts seem, most often, to be a sequel of "Isoniazid" therapy. Endogenous spread of tuberculosis occurred during therapy in five patients despite the fact that "Isoniazid" resistant organisms are less pathogenic in the guinea-pig. Patients with fully resistant organisms also had the most extensive and destructive disease.

Mechanism of Death in Rheumatic Heart Disease.

J. B. WALLACH, L. LUKASH AND A. A. ANGRIST (*Am. J. Clin. Path.*, April, 1956) in a series of 8676 autopsies found 509 examples of rheumatic heart disease. In each case, the relation of rheumatic heart disease to the death of the patient was classified as incidental, contributory or directly causal. The results indicate that with increasing age rheumatic heart disease is more often a relatively inactive, incidental lesion, and its role in the cause of death becomes less significant. Active rheumatic disease was significant as a cause of death in the first two decades of life, but in middle age heart failure due to inactive rheumatic disease was often the cause. Bacterial endocarditis was most common in the second and third decades; after this the incidence fell. However, a rising incidence was found after the age of sixty years. Embolic phenomena caused most deaths in patients aged from forty to seventy-nine years when factors, other than rheumatic activity, prevailed in the formation of thrombi. In the elderly, the occurrence of senile non-specific endocarditis is invoked as a significant factor in the causation of emboli.

Wegener's Granulomatosis.

E. W. WALTON AND P. O. LEGGATT (*J. Clin. Path.*, February, 1956) have reported a further case of Wegener's granulomatosis which is characterized by (i) necrotizing granulomatous lesions in the upper air passages, or in the lungs, or both, (ii) generalized focal necrotizing angiitis, and (iii) glomerulitis, in which there are thrombonecrosis of tufts, capsular adhesions, and gradual conversion into a granulomatous lesion. The case described is that of a forty-two year old woman who was thought to be suffering from pulmonary tuberculosis. Her condition improved with a short course of PAS and streptomycin. The subsequent administration of streptomycin was accompanied by a violent sensitivity reaction. Several attempts were made to desensitize the patient, but each time she suffered a violent reaction and on each occasion a further manifestation of Wegener's granulomatosis appeared. After ten months the patient died. At autopsy the various features of Wegener's granulomatosis were recognized. As the result of this case, the authors postulate that there occurs an initial lesion in the respiratory system, and that a state of hypersensitivity follows during immunization to bacterial or tissue breakdown products or, as in this instance, as a result of drug therapy. In this case, as in the others previously reported, respiratory symptoms occurred first.

Mediastinal Cysts.

M. A. ABELL (*Arch. Path.*, May, 1956) has made a review of the cysts and neoplasms removed from the mediastinum in a fifteen year period at the University Hospital, Michigan. There were 133 primary neoplasms and cysts, of which the 43 neurogenic tumours comprised

the largest group. There were 36 primary cysts, 19 thymic tumours, 14 teratomata, eight tumours of connective tissue or of vascular origin, six lymphoblastic tumours and seven miscellaneous neoplasms. The cysts form the material of this review; there were 17 tracheo-bronchial, one tracheo-œsophageal, three œsophageal, four gastrogenous, eight pericardial cœlomic, two thymogenous and one meningeal. The tracheo-bronchogenous cysts were the most common and were found at ages ranging from seventeen to fifty-six years. Ten were from male and seven from female patients. Pressure symptoms were produced in five patients. Two cysts were incidental findings, and were observed and removed during operations for other lesions. The remainder were detected on radiographic surveys. Although arising in the mid-mediastinum, cysts of this type usually presented as posterior mediastinal tumours. The structure of these cysts recalled the structure of normal trachea. They are thought to be congenital and to develop through sequestration of a group of cells from the laryngo-tracheal groove. They may represent the abnormal development of supernumerary lungs early in embryonic life. The one tracheo-œsophageal cyst in this series was in a child seven and a half months of age. The cyst was in the superior mediastinum. It was probably created during the early formation of the tracheal groove. This would account for the presence of both respiratory and œsophageal elements. Two of the œsophageal cysts were in the superior mediastinum and were intimately related to the œsophagus. In one patient there was also a separate bronchogenous cyst. The third cyst was an incidental finding in the repair of a tracheo-œsophageal fistula in a three-day-old child. Two of these cysts are thought to represent pinched off diverticula. They had striated muscle in their walls. The third cyst seemed to be related to the development of a tracheo-œsophageal fistula. This type may be lined by ciliated epithelium and represents the fœtal type of œsophageal mucosa. The gastro-enterogenous cysts were all from boys aged from four months to five years. All were in the anterior mediastinum and they produced symptoms by pressure. In all patients there were abnormalities of the cervical or dorsal vertebræ. In three instances there was a lack of spinal fusion, which resulted in hemivertebræ, and in one there was a congenita mid-dorsal scoliosis. The cyst was attached by a pedicle to the vertebral column in each case; in one patient there was also a duplication of the terminal part of the ileum. Three of the cysts contained gastric mucosa. The other had lost most of its lining; that which remained consisted of a single layer of columnar cells. Pericardial cœlomic cysts were found in patients aged from thirty-two to forty-nine years. Only four patients had symptoms and, in the other four patients, the cysts were incidental findings during a radiological survey. These cysts appear to be formed during development of the pericardial cavity and most of them have some form of attachment to the pericardium. Thymogenous cysts occurred in the anterior mediastinum; the two examples in this series were from women aged forty-five to fifty years. One of these cysts caused symptoms by pressure. These cysts may be very large. They are thought to be derived from persistent elements of the thymo-pharyngeal duct. The one example of a meningocele in this series presented through a defect in the spinal column. The patient also had some of the stigmata of neurofibromatosis, as occurs in most of the reported cases.

MORPHOLOGY.

Innervation of Human Dentine.

R. COCKER AND J. M. HATTON (*J. Anat.*, April, 1955) state that by the combined use of slow decalcification and a modified silver nitrate staining technique, they have confirmed the course of nerve axons in the pulp and predentine of human teeth. In addition, they have demonstrated a small number of axons penetrating into the calcified dentine of human teeth.

Perivascular Spaces of the Central Nervous System.

D. H. M. WOOLLAM AND J. W. MILLEN (*J. Anat.*, April, 1955) state that, because of the complexity of the tissue layers involved and the difficulty in obtaining a suitable indicator which, when injected into the subarachnoid space, serves to outline the perivascular spaces, the published accounts of these spaces are confused and contradictory. In the present investigation, two groups of experiments were performed. In the first group, the single injection of indian ink into the subarachnoid space of the adult rat was followed two hours later by the administration of hypertonic saline by the intravenous route. In the second group the daily injection into the subarachnoid space of colloidal carbon in the newborn rat from birth to about three weeks of age served to outline the perivascular spaces. In both groups the brain and spinal cords were removed, fixed, sectioned, stained and examined histologically. It was found that there are two spaces surrounding the blood vessel in the central nervous system. The first is the true perivascular space which communicates with the subarachnoid space, but not with the perineuronal spaces. External to this true perivascular space lies an artefact space which communicates with the perineuronal spaces and with the epispinal space of His between the *pia mater* and the surface of the brain and spinal cord, but not with the subarachnoid space. All these spaces are artefacts, the results of shrinkage consequent to fixation and sectioning.

Arterial Supply of Prostate and Seminal Vesicles.

E. J. CLEGG (*J. Anat.*, April, 1955) reports that in a total of 21 pelvic halves the blood supply to the prostate gland and seminal vesicle was studied both by dissection and radiologically. A definitive prostatic artery was found in all cases examined; it was the most constant branch of the prostato-vesical artery. The superior rectal artery was found to supply the gland in 32·1% of cases, a much higher figure than that of Awataguti (1939). The vesiculo-deferential artery was found to supply the seminal vesicle in all cases through its anterior vesicular branch (author's terminology). In eight cases out of 15 the posterior vesicular artery (author's terminology) was a branch of the prostato-vesical artery, and in six cases a branch of the gluteo-pudendal trunk. It is considered that variations in the nomenclature of blood vessels in this region account, in the main, for the wide diversity of findings in the blood supply of the prostate and seminal vesicles.

Rectal Valves of Houston.

P. H. S. SILVER (*J. Anat.*, April, 1955) states that a review of the literature concerning Houston's valves in the adult rectum has shown that there is disagreement on many material anatomical points. This is true regarding both the position and number of valves, and the degree to which the layers of the rectal wall are incorporated into the valves. As far as can be discovered, the valves have never previously been studied specifically in the embryo and fœtus. The author reports observations he has made in 21 human embryos and fœtuses. The valves make their appearance towards the end of the third month; the submucosa becomes thickened and is invaded successively by the circular and longitudinal muscle coats and finally by the perirectal connective tissue. This is a consistent and reliable picture. The anterior and posterior parts of the longitudinal muscle coats play no part either in the production or in the maintenance of the valves. When seen from outside, the rectum usually appears straight or almost straight, but this belies the nature of the lumen, which zigzags past the valves of Houston, because they project from the lateral walls like horizontal baffle plates. When the rectum is distended with meconium the valves are taken up into the rectal wall.

Renal Glomerular Efferent Vessels.

J. P. SMITH (*J. Anat.*, April, 1956) has studied blocks from 21 adult human normal kidneys and reports that the efferent vessel of the human renal glomerulus may be a muscular arteriole or a solitary wide endothelial tube, or it may consist of numerous capillary vessels. Occasionally, more than one efferent vessel emerges from a single glomerulus. Rarely, efferent vessels emerge through Bowman's capsule at a point remote from the hilum of the glomerulus.

Nodes of Ranvier in the Central Nervous System.

O. C. PEASE (*J. Comp. Neurol.*, August, 1955) states that there has remained a persistent doubt in the minds of many authors that the nodes of Ranvier exist centrally, and, if so, whether they are really identical with peripheral nodes. In the present investigation, here reported, he states that unmistakable nodes were frequently found. They resemble peripheral nodes, although perhaps there is a somewhat larger gap in the myelin. Their elusiveness in the past is attributed to the difficulties of obtaining good cytological fixation in the central nervous system.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on April 17, 1956, DR. J. E. REIMER, the Assistant Medical Superintendent, in the chair. The principal speaker was DR. B. P. BILLINGTON, an honorary assistant physician of the hospital.

The following clinical history was presented.

Clinical History.

The patient, a young male school teacher, aged twenty-four years, on his admission to hospital was lethargic and unable to give an account of his illness. From his brother it was learnt that he had suffered a gradual loss of consciousness commencing about four hours previously. Earlier that morning he had seemed normal except for an apparent slight weariness and slowness of speech. Gradually his speech had become more confused, and he had begun to repeat the same phrases. At 12 noon he lost consciousness, and had broken out into a heavy sweat. There were no twitchings or convulsions, and he was continent of urine and fæces. His past history went back two years, when he had collapsed at home one day. He was taken to hospital, where his condition was provisionally diagnosed as epilepsy or aneurysm. He was given "Dilantin", and suffered no further attacks for a year. Then he commenced to have attacks with tonic and clonic phases, and, more commonly, periods when he lapsed into a deep sleep, lasting from two to twelve hours, from which he could not be aroused. Sometimes he would awake normally, at other times after a "twitching attack". Physical examination and air studies at this time revealed no abnormality, but encephalographic examination revealed an abnormality consistent with epilepsy. "Mysolin" therapy was instituted, but he still suffered occasional "sleeping attacks". He had not complained of headaches, of vomiting, of faulty vision, or of any other symptoms. His habits were moderate, except that he smoked 30 cigarettes per day. There was no family history of epilepsy, and his only previous illness was acute appendicitis at the age of eight years. His mother subsequently said that he had suddenly gained two stone in weight during the last twelve months.

Examination of the patient revealed him to be a cold, sweating, limp, comatose young man in a good nutritional state. No other abnormality was noted except a bilateral Babinski response. His blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic, his pulse rate was 80 per minute and his temperature was 98° F.

The patient remained in the state described for some hours, but the following day gradually recovered and became reasonably well orientated. The next day the nurse observed the patient having a "fit" lasting about thirty seconds, during which he passed urine. Examination of the patient after this episode revealed him to be comatose, with bilateral extensor plantar reflexes, some facial twitching and spastic lower limbs. Thus the patient progressed, occasionally having convulsive seizures or alternatively passing into a deep sleep, with at other times periods of relatively clear consciousness. One week after his admission to hospital he began to have an elevation of temperature up to 102° F., and for several days never below 99·8° F. He was given penicillin despite the absence of localizing signs. During this phase he was responding only to painful stimuli, was sweating profusely and was thought to have slight neck stiffness. His state of consciousness remained the same, and three days after the onset of the fever he developed extensive vascular blebs on the trunk, face and arms with some hæmorrhagic spots on his arms. He was now being fed by a Ryle's tube. A tentative diagnosis of chickenpox with encephalitis was made. The temperature dropped to normal the day after the vascular eruption appeared. A pneumoencephalographic examination revealed a diffuse enlargement of the ventricular system and a large amount of air present in the subarachnoid space suggestive of encephalitis.

The patient continued in a semi-dazed state, occasionally taking fluids by mouth, but never regaining full consciousness and sometimes lapsing into coma or convulsions. One week after the pneumoencephalographic examination he suffered a "fit" in the left side of the body; this was followed by a somnolent state. The seizure was stated to have lasted ten minutes, and the right pupil was thought to be larger than the left. After this the patient improved considerably. He was able to sit up in a chair and take an interest in the newspaper, and his general condition improved. However, one week later he suffered a series of head twitchings and excessive salivation, culminating in a convulsive seizure with cyanosis. He was thought to have aspirated some vomitus. He was resuscitated, but his condition deteriorated progressively. He developed pronounced respiratory distress and tachycardia (rate 160 per minute), and he became pale and sweating. The respiratory rate fell from 40 to 20 per minute in a few hours, his condition deteriorated, and he died forty-eight hours after this last seizure.

A number of special investigations were carried out with the following results. X-ray examination of the chest and skull revealed no abnormalities. A blood examination gave the following information: the hæmoglobin value was 14·3 grammes *per centum* and the leucocytes and platelets were normal. The Wassermann and Kahn tests produced negative results. The non-fasting blood sugar content estimated on various occasions was 141, 55, 202, 294 milligrammes per 100 millilitres. The serum calcium content was 10·8 milligrammes per 100 millilitres. The cerebro-spinal fluid pressure was 200 millimetres; the fluid was clear, and responded freely to pressure variations. The fluid contained, per 100 millilitres, 700 milligrammes of sodium chloride, 20 milligrammes of protein and 80 milligrammes of glucose; it contained no cells.

Clinical Discussion.

DR. B. P. BILLINGTON: I say right at the outset that I do not know the cause of this young man's fatal illness. However, it will be interesting from the point of view of discussion to consider some of the possibilities.

It would appear to me that when he died no definite diagnosis had been made, and in general it appears as though the trouble lay in the nervous system. I will deal with the problem rather from the point of view of the general physician, not attempting to be involved in neurological intricacies.

In summary, the case is that of a man of twenty-four with a two-year history, intermittent at first, of funny turns, fits, coma and finally death.

The first possible diagnosis we must raise is that of idiopathic epilepsy. The story was episodic, the fits had tonic and clonic phases, the electroencephalogram showed an abnormality consistent with such a diagnosis, and earlier there had been some response to antiepileptic drugs. But on the other hand a man of twenty-four developed epilepsy, if it were so, for the first time, without a positive family history, and died of it in two years; this is not the course of idiopathic epilepsy, and I exclude it on these grounds as the correct diagnosis. Admittedly, if it were, I doubt whether this case would be a suitable one for presentation at a clinico-pathological conference.

A similar set of arguments will exclude idiopathic narcolepsy, which might have been suggested by the history of sleeping attacks and gain in weight. We have too many other disturbances to implicate idiopathic narcolepsy as the cause of this fatal illness.

The history seems to point to an organic intracranial lesion, but I think we should first consider some general disorders which may be associated with funny turns and fits.

Firstly, this is not a case of hypertensive encephalopathy; there was no evidence of hypertension. Nor is there a suspicion that the episodes were Stokes-Adams attacks, as there was nothing to suggest cardio-vascular disease, nor were there any obvious cardio-vascular abnormalities found on examination in hospital at the time of the attacks, such as slow pulse or arrhythmia.

We might mention here the possibility of the patient having a phæochromocytoma, in which disorder there may be paroxysmal attacks of a peculiar nature, usually with hypertension; but certainly in some instances the hypertension may not be a prominent feature, and the clinical signs of sweating and prostration may be dominant, and in some cases paroxysmal tachycardia may be more noticeable than hypertension. In this case there does not appear to be enough evidence to implicate a phæochromocytoma as a basic cause.

Irritability of the carotid sinus can also produce peculiar paroxysmal attacks, but here we have no history of slowing of the pulse or fall in blood pressure, which are usually associated with the carotid sinus syndrome.

Another possibility to consider among the general causes of fits and funny turns is that of a general intoxication; the only theoretical possibility here is that of lead encephalo-

pathy. We have no real evidence to implicate lead, as there was no contact history, no lead line, no stippled cells, and, moreover, the attacks were very sporadic at the outset of the illness.

Another cause of convulsions, taken from the general point of view, would be hypoparathyroidism, but I think we can rule this out because there is no history of numbness and tingling before the attacks and no evidence of tetany; the patient had not undergone an operation upon his thyroid gland; we have none of the stigmata of primary hypoparathyroidism, and, moreover, the serum calcium level was normal.

One further possibility to exclude as a general cause of fits and funny turns is that of hypoglycæmia; we are told that this man had fits on one hand and attacks of sleepiness and drowsiness on the other, and on examination in most of these attacks he was limp and sweated profusely. These clinical manifestations fit very well if we postulate hypoglycæmia as a basic cause; but we are not told whether the attacks were precipitated by fasting, or in fact whether there were any precipitating factors at all. Apparently this possibility had been considered because, as you will see in the protocol, some blood sugar estimates had been done; although taken at random, they gave figures which vary from a low figure of 55 milligrammes per 100 millilitres to a high figure of 294 milligrammes per 100 millilitres. I do not wish to consider this possibility in any more detail at the moment; we will return to it later. Nevertheless, hypoglycæmia as a diagnosis in this patient certainly has points in its favour from the clinical side if not from the laboratory side.

I think we can defer no longer an attack upon the cranium to discover there some possible causes for this man's illness.

Two diagnoses were mentioned in the protocol—namely, aneurysm and encephalitis. Referring to aneurysm, perhaps the initial attack may have been construed in this way, but the subsequent course of events makes this a somewhat unlikely diagnosis. There were no focal signs or evidence of meningism except possibly towards the end, and the cerebrospinal fluid at all times was apparently quite clear, without increase in protein, without red cells or xanthochromia. How could a ruptured aneurysm cause generalized epileptic fits? Only, I think, by a severe leak into the subarachnoid space or into the ventricular system. In this case the spinal fluid was normal, so it would appear that we can rule out an intracranial aneurysm as the cause of this disorder.

Encephalitis was mentioned; we have towards the end of this illness a febrile episode with a skin rash which was diagnosed as chickenpox. Perhaps this patient did, in fact, have chickenpox; I can find no real way of integrating the febrile illness and skin rash with the rest of the story, although probably the pathologist will show me where I go astray. I do not think that the label of encephalitis will fit this case, with its episodic progress over two years and yet apparent normality in the intervals. At the same time, there is no evidence of a meningo-encephalitis because, except for one episode of questionable neck rigidity, there is no clinical or pathological evidence to suggest that the meninges were involved. Also, against a chronic meningitis is the fact that the illness was episodic and, moreover, good ventricular filling was obtained on the pneumoencephalogram with air introduced into the subarachnoid space by the lumbar route, an unlikely event in any chronic intracranial meningitis.

The possibility of a form of cerebro-spinal syphilis must be considered. I can think of no specific type of syphilis, despite its protean manifestations, which could give quite the same story as the man presented; the blood Wassermann was negative and the cerebro-spinal fluid findings as far as they are given were normal. Could the rash at the end have been a manifestation of secondary syphilis? I think it is unlikely. I will admit I have seen a peculiar week-end case of a young man being admitted with fits and coma, and dying within thirty-six hours in which the diagnosis was secondary syphilitic encephalitis, but in the present case we are dealing with a case of some duration. Surely the story suggests some progressively expanding intracranial lesion, with peculiar episodes. We must consider this possibility in detail. Pathologically speaking, what types of lesion could give rise to the picture in this man's case? Cerebral tumour, cerebral abscess and subdural hæmatoma are possibilities. I think cerebral abscess is unlikely because there is no history or clinical evidence of a focus from which a cerebral abscess could have arisen, or of a previous pyæmia, and, moreover, the cerebro-spinal fluid was normal. The episodic history strongly suggests a subdural hæmatoma with its characteristic lucid and normal intervals alternating with fits and funny turns. However, against this there is no history of head injury, or radiological evidence that the skull may have been fractured, there were no localizing symptoms or signs, the electroencephalogram and the pneumoencephalogram did not indicate its location, and the cerebro-spinal fluid was normal.

If we attempt to implicate a variety of cerebral tumour, vascular or otherwise, we find two features of the illness which are peculiar—namely, an absence of symptoms and signs of raised intracranial tension, and an absence of localizing features. There is no history of vomiting or headache, there was no papilœdema, the cerebro-spinal fluid pressure was normal, and its protein content was not increased.

In attempting to localize an intracranial space-occupying lesion let us consider the cerebral hemispheres first. There is no suggestion of the fits being focal at any stage—rather do the tests tend to indicate a diffuse process involving both hemispheres, as shown by the diffuse abnormality in the electroencephalogram and the failure to find shifting of the ventricular system in the pneumoencephalogram. I can find no evidence to localize a space-occupying lesion within the cerebral hemispheres, nor indeed do I think the evidence can suggest such a lesion below the tentorium, where characteristically the symptoms and signs of raised intracranial tension should occur early and be prominent. What about mid-line lesions? If we consider a lesion in the brain stem, we have to explain *grand mal* attacks, the absence of localizing signs from the point of view of the cranial nerves, which are almost invariably affected by lesions in this region, and the normal cerebro-spinal fluid. It would appear, therefore, that the possibility of a space-occupying lesion in the brain stem is not admissible. What about the possibility of a third ventricle tumour? This is certainly a possibility; but these lesions tend to cause some internal hydrocephalus, which appears to be absent historically in this case, and, moreover, we would not expect to be able to show the two main ventricles by pneumoencephalography via the lumbar route.

A final locus within the cranium remains to be considered—namely, the area between the pituitary and the hypothalamus. We have certain features which suggest involvement of this area—namely, a history of attacks of somnolence, of weight gain, and possibly a disturbance in carbohydrate metabolism; but we then have to explain the typical attacks of *grand mal* and the absence of disturbance in ocular innervation, visual acuity or visual field disturbance which occurs with lesions in this area. Further, there was no radiological abnormality such as calcification of a tumour of Rathke's pouch, erosion of the clinoid processes or enlargement of the sella. We know, moreover, that disturbances of hypothalamic and pituitary function are commonly false localizing signs in intracranial diseases of some duration.

So far we have not been able to find real evidence to support a diagnosis of a space-occupying lesion in the cranium. Nor have we been able to localize such a hypothetical lesion. The evidence points surely to a form of diffuse intracranial disturbance, involving both hemispheres at least. If we examine the findings at pneumoencephalography, we find an excess of air both within the ventricles and outside the brain, in the subarachnoid space. This suggests to me, not a type of hydrocephalus, but some sort of cerebral atrophy, a condition which would also fit the electroencephalographic findings of a diffuse disturbance of brain activity.

The commonest cause of cerebral atrophy taken as a whole must be arteriosclerosis; but surely we cannot postulate that this was the cause in a young man of twenty-four without other evidence of vascular degeneration. Nor indeed at this age can we postulate that the cerebral atrophy, if present, was due to a congenital lesion, a porencephaly. Demyelinating diseases may cause at some stage shrinkage of brain substance; certainly disseminated sclerosis and Devic's disease do not fit the clinical picture. Schilder's disease remains a theoretical possibility, but I do not think there is evidence to proceed further on this line of thought.

Certain cerebral abiotrophies can cause atrophy of brain substance, and some of these may become manifest later in life as variants of the tuberous sclerosis complex, and may present with general epileptic convulsions; but we have here no evidence of the usually associated skin or retinal lesions or of involvement of other viscera; the possibility of the cerebral atrophy in this case being due to an abiotrophic process is unlikekly.

So far, we have not reached any definite conclusions, and we might mention a few rare possibilities. Cysticercosis

occasionaly causes fits in young people, but here there is no evidence of intracranial calcification. Torulosis is another possible cause of mysterious progressive disease of the central nervous system, usually presenting in the manner of an expanding intracranial lesion, but with definite biochemical abnormalities in the cerebro-spinal fluid, even if the organisms are not specifically sought, abnormalities which are not present in this case.

Occasionally there are some cases of cerebral atrophy of unknown cause, often with a pathological picture of diffuse gliosis. I can remember having seen such a case demonstrated in a young man with progressive mental loss and fits; the autopsy showed a diffuse gliosis with cerebral atrophy, but no evidence of a primary cause. Could this have been the case here? I think, as we have not been happy with any other intracranial diagnosis, that it probably fits best, but I would have anticipated that in addition to the fits we might have had a story of progressive mental deterioration, and according to the protocol this does not appear to have been so.

We have not yet made a confident diagnosis. I am loath to do so, but two currents cross—namely, hypoglycæmia and cerebral atrophy. Severe prolonged hypoglycæmia can cause organic changes in the brain, with gliosis and I presume cerebral atrophy, although I have not seen such a case.

This appears to be the only point at which the tangled skein makes some sort of pattern. If, however, we are to postulate that this was a hypoglycæmic illness, what have we to support it? Certainly the clinical description of the attacks fit, but we have only one solitary random blood sugar figure of 55 milligrammes per 100 millilitres, and this is somewhat inadequate upon which to make a confident diagnosis of hypoglycæmia.

If this was hypoglycæmia, and a fatal spontaneous hypoglycæmia at that, the only organic diagnosis that can be deduced is the presence of an insulin-secreting tumour of the β cells of the islets of Langerhans of the pancreas, either in the normal situation of the pancreas or in ectopic pancreatic tissue elsewhere. Certainly these cases are uncommon and may present in most peculiar ways, and if undiagnosed may result in death usually in coma or after gross cerebral deterioration.

In searching for an answer to this case I looked up a review of insulin-secreting tumours to ascertain what levels of blood sugar are consistent with this diagnosis, and in a review of 160 cases, only six showed a minimum blood sugar value of over 50 milligrammes per 100 millilitres.

In the face of this evidence the possibility of the present case being one of islet cell tumour is more remote, particularly when we try to explain the blood sugar value, also random, of 294 milligrammes per 100 millilitres. It would appear, therefore, for want of any other explanation, that this man had a disturbance of carbohydrate metabolism, and taken with somnolence and weight gain, we are forced back to the hypothalamus as a possible site of the lesion, but this will not, to my mind, explain the presence of *grand mal* attacks.

I can say no more about this case; I do not know what was the matter with the patient, but I would like the question of hypoglycæmia answered once and for all. I wonder whether Dr. Wardlaw might like to comment, and particularly to answer the question as to whether he would consider these random blood sugar findings consistent with a diagnosis of hypoglycæmia.

DR. H. S. H. WARDLAW: There were other measurements of sugar. As I interpret the figures, those looking after the patient considered the possibility of hypoglycæmia and had a blood sugar done which showed a low figure of 55 milligrammes per 100 millilitres. Measures were probably then instituted to cope with the hypoglycæmia, and these, according to the subsequent figures of 202 and 297 milligrammes per 100 millilitres, were only too effective. But, according to our information, the condition of the patient was not improved. It would seem possible that interest then shifted to the central nervous system and measures for dealing with hypoglycæmia were discontinued, so that the blood sugar began to fall, as is reflected in the cerebro-spinal fluid sugar figures of 120, 85 and then 30 milligrammes per 100 millilitres. It seems a pity that simultaneous measurements in the blood and cerebro-spinal fluid were not made more frequently.

DR. W. EVANS: Dr. Billington I think has excluded most of the local causes—epilepsy, encephalitis, and also most of the general causes of convulsions. Although the suggestion of a hypoglycæmia due to a pancreatic disorder is made, I favour some cerebral condition because of the fact that signs and symptoms throughout point to a cerebral condition, and electroencephalography has shown abnormalities and the pneumoencephalogram has shown enlargement of the ventricular system. In the young adult with a history of fits, one must always very strongly suspect a cerebral tumour even if one cannot confirm any localizing signs. I think particularly all of us have made errors of missing a cerebral tumour in a young man of this age who has come complaining of epileptic fits, and I would think the probable suggestion is a cerebral tumour somewhere in the region of the hypothalamus. That would affect his carbohydrate metabolism. The other things which were of interest to me, and have not been dealt with particularly, were the fever and those hæmorrhagic manifestations on his trunk and arms. I wondered whether it was an incidental thing, such as varicella, as was suggested, but it did raise the the question for a moment as to whether he had a periarteritis or something of that type, but I think the major symptoms point to a cerebral tumour.

Another possibility, too, a rare possibility, mentioned by Dr. Billington, was torulosis, which always has this vague history finally leading to death; but the cerebro-spinal fluid examination was normal, so that in my opinion the preponderance of evidence is in favour of cerebral tumour.

DR. C. HUDSON: I have not anything very coherent to add, but I have made some jottings.

In the investigations that were made, the electroencephalogram comes into my department, and I would like to say that the electroencephalographic report is rather vague here, as it often is, and the reason for that, of course, is that generalized abnormalities are present, and this may occur in a number of diseases. One of the diseases in which such a state of affairs exists is idiopathic epilepsy in the interseizural record; but other things such as raised intracranial pressure, encephalitis, myxœdema and hypoglycæmia may give a record which shows generalized mild abnormalities, and we often cannot be very specific about it.

So the fact that this report supported the diagnosis of idiopathic epilepsy does not really mean very much. I feel that this youth must have had a lesion somewhere in the vicinity of the hypothalamus. I do not quite agree with Dr. Billington in his statement that it is in this area, because diencephalic epilepsy or ventricular epilepsy as it is sometimes called does occur with lesions of this nature, and they are associated with tachycardia, fever, sweating, flushing and disturbances typical of a hypothalamic disorder, and they may be followed by generalized convulsions and coma. So I think that the fits are consistent with hypothalamic tumour.

This vesicular rash might, I stress might, also fit in with a hypothalamic sort of vasomotor disorder; but I admit this is a difficult one. I shall return to that in a moment.

In the differential diagnosis that was given in the paper, aneurysm was mentioned, and as Dr. Billington says, aneurysm is not a very common cause of generalized fits; but angioma, cerebral angioma, is, and this should be considered.

Dr. Evans mentioned periarteritis. A collagen disease of the *lupus erythematosus* type may give rise to focal and generalized cerebral lesions, and might also give rise to vesicular blebs, and I think that we should consider these.

This problem of cortical or cerebral atrophy that Dr. Billington mentioned is, I think, a very interesting problem. Cerebral atrophy does occur in people when you would not expect it, in quite young people. It may be localized or generalized and it may cause fits. We have seen this frequently in young people as a cause of Jacksonian epilepsy with definite focal features in the electroencephalogram, and we have been unable to account for the atrophy, and it is frequently not associated with any mental impairment. In fact, the ones we see usually have no mention of impairment.

I have not actually seen a case in which a person with cortical atrophy presented with hypothalamic involvement. You could have hypothalamic involvement. That brings us down to what type of lesion we are dealing with. I think that a tumour in that area would be the most likely diagnosis. A large tumour I think would produce more signs of raised intracranial pressure, and I am afraid I could not make a statement as to what pathological lesion is present; but I think there is a lesion of a degenerative nature or a small neoplastic lesion in the hypothalamic area.

DR. T. I. ROBERTSON: I think that the history points very strongly to the hypothalamus. Dr. Hudson has covered the field extremely well, indicating just which signs do so. It seems to me that the sequence has probably been hypothalamic tumour and either diencephalic epilepsy with

generalized fits or hypoglycæmia causing the fits. It is necessary to elucidate the nature of the lesion. I would think a small, probably infiltrative tumour the most likely, but would also mention the possibility of tuberculoma.

DR. P. HALL: The one diagnosis so far suggested which involves the endocrine glands is that of islet cell tumour. I think one can only conclude from the clinical and laboratory data available that this diagnosis, though not definitely excluded, receives no positive support. Dr. Wardlaw has presented the blood and cerebro-spinal fluid glucose levels in such a way as to suggest that hypoglycæmia may well have been present. However, the only really consistent disturbance in carbohydrate metabolism found in islet cell tumours is a failure of the blood sugar to maintain normal levels with prolonged starvation. Fasting sugar levels are variable, and random readings (such as those presented) do not exclude the possibility of islet cell tumours.

A lesion of the hypothalamus has also been suggested, and this seems to me the most likely diagnosis so far presented.

PROFESSOR F. R. MAGAREY: Dr. Billington told us that there was no evidence of papilœdema. I cannot find it on my sheet. Is there any record of the disks being examined?

DR. F. CROLL: I would like to mention that this patient developed his rash after penicillin therapy and that the penicillin may have been the cause.

Dr. Billington: In answer to Professor Magarey, I took the record in the protocol of the absence of significant physical abnormalities in the nervous system to exclude the presence of papilœdema. It would appear from the discussion that the consensus of opinion of the group is that we expect to be shown a lesion in the hypothalamus, although we cannot infer its nature.

Autopsy Report.

The following autopsy report was given by DR. E. HIRST.

The patient had been a thin young adult, six feet in height and weighing 125 pounds.

The significant gross findings were as follows.

There was patchy consolidation of the lungs suggestive of bronchopneumonia. A small ovoid tumour (two centimetres by one centimetre by one centimetre) was present in the tail of the pancreas. A small subdural collection of blood was found in the left anterior and middle cranial fossæ. The brain appeared grossly normal.

Microscopic examination confirmed that the growth in the tail of the pancreas was an islet cell tumour. However, two microscopic tumours were present in the head. In addition there was generalized hyperplasia of islet tissue throughout the pancreas.

There was generalized vascular congestion of the brain. Small petechial perivascular hæmorrhages were found in scattered areas. Widespread areas of neuronal damage were revealed by honeycombing and disappearance and degeneration of nerve cells. There were foci of glial proliferation and satellite glial cells in the cerebral cortex and basal ganglia, also indicating cerebral degenerative changes.

Dr. Billington: When I entered this room, I had made up my mind that if I was forced, unwillingly, to make a diagnosis, I would say that the patient probably had a pancreatic islet cell tumour with secondary cerebral gliosis and atrophy. I was supported by Dr. Wardlaw's revelations, but after hearing Dr. Hudson's eloquent address, I changed my mind. I realize now that I should not have done so. If this points a moral, surely it is that if one has an opinion one should adhere to it.

I think that this has been a very interesting and stimulating case, and I am sure it teaches a lesson that a general approach to what appears to be strictly a neurological case can bring fruitful results. I shall be very glad to hear what the other speakers think, particularly whether they are surprised by the outcome or not.

DR. K. B. NOAD: I think that it is a very good thing in these clinico-pathological conferences to have an epilogue. Canio in the prologue to *I Pagliacci* sings: *"Io sono il prologo."* So perhaps I am the epilogue.

This case was a very difficult diagnostic problem, as is obvious from the protocol and the discussion. In the beginning we were taken up with this idea of a hypothalamic disturbance, and we got in touch with the patient's medical attendants in Queensland, people at the Brisbane General Hospital, who had gone into it carefully, and they were convinced that he was an epileptic, and we wondered whether these could be curious post-epileptic comatose states. Of course, the most curious things can happen in epilepsy, as you know. However, where did we go wrong? We went wrong partly because we had bad luck. Now if we had only put down the things the way Dr. Wardlaw has done, it would have been obvious that the man had hypoglycæmia. He had all the features of hypoglycæmia, the coma, the sweating, the pallor, and at times peculiarities of behaviour, irritability, sometimes focal neurological fits, sometimes generalized fits, all very characteristic of hypoglycæmia. But we had bad luck, because I had a very excellent registrar, and the blood sugar readings of 202 and 294 were the result of his efforts with intravenous glucose; but unhappily it did not improve the man's condition. Even his coma did not significantly improve with the glucose he was given, and that, of course, threw us off the scent. What we should have done was to starve him for twelve hours and then do his blood sugar. One should always do this in these patients, give them nothing for twelve hours, and, as Whipple points out, you will almost always find that their blood sugars will fall to 40 or even 20 milligrammes per 100 millilitres, and that is another reason why we went wrong.

Islet cell tumour is very like phæochromocytoma—sometimes the blood pressure is normal in phæochromocytoma. It is pushing out the pressor substances sometimes, and sometimes it is not, and apparently the same sort of thing happens in these tumours because occasionally the blood sugars are found to be normal in these patients. But if they are starved, then usually the blood sugar is found to be very low, even under 20 milligrammes per 100 millilitres. I think that this case is a most fascinating one. Of course, the autopsy findings show that he really had a hyperplasia of the islet cells as well as this tumour formation. It would have been very difficult to do anything for this man. I have had one of these patients who was in this hospital at one time and Mr. Starr resected the pancreas up to the superior mesenteric vessels, as is advised, but it made no difference. This woman had focal attacks and peculiarities of behaviour and all the rest of it, so we went in again and took the remainder of the body of the pancreas, leaving only the head. She has been amazingly well and it has revolutionized her life.

DR. H. M. WHYTE: I should like to add a couple of points now that we can be wise after this event. One is that there has been some mention lately of confusion between barbiturate poisoning and this condition. Many of the clinical features of hypoglycæmia were found to occur in barbiturate poisoning, and this led to an examination of carbohydrate metabolism. It was found that barbiturates did disturb carbohydrate metabolism, but in the opposite direction, in so far as blood sugar levels are concerned. This, of course, does not mean to say that the glucose was being used normally. Vitamin *B* was said to reverse these changes in blood sugar and blood pyruvate.

A second thing: even if hypoglycæmia is found—and I think the point Dr. Noad has mentioned should be stressed, about prolonged fasting with serial observations going on for twenty-four or forty-eight hours to get the answer—it should be remembered that conditions other than islet cell tumours might cause it. Conditions have been described and recently reviewed,[1] in which masses have been present in the abdomen, and significant hypoglycæmia has been present, which has been relieved by the removal of these tumours, and these have turned out to be such things as retroperitoneal sarcomas, fibromas and pseudomyxomatous cysts. In these cases the pancreas has been absolutely normal, and the patients have been normal after removal of the tumours.

Another point worth mentioning is what was brought out by Richardson and Russell in 1952, reviewing six cases like this, especially their first case, which was a person who took five months to go into coma and then died two months later. They, too, found this pathological change with disappearance of neurons; and, what is more, they stressed, as a result of their experience, that the blood sugar is not necessarily low and that there may be no response to sugar; in fact, they were describing just this case.

There is one other point that I would like to mention and invite comments upon, and that is the question of chickenpox that this man got, and which is something to add to the confusion of the moment. I remember the registrar looking into it very carefully at that time and finding that no cases of encephalitis had been reported as being due to chickenpox and occurring before the rash, as might have been postulated in this man. But last year Lander, from

[1] *Lancet* (1956), 1: 304.

South Australia, reported acute hæmorrhagic leucoencephalitis due to chickenpox in a man who died. He had chickenpox, developed cerebral signs five days later and died a few days after that. I wondered whether the findings in this sort of disease resemble what was found here—whether he sustained cerebral damage from chickenpox in addition to what was caused by the hypoglycæmia.

DR. NOAD: I thought that he did have chickenpox. Of course, as Dr. Croll mentioned, it is quite a possibility that it was a penicillin reaction, but most unlike any of the penicillin reactions we had ever seen, and it was very like chickenpox. So in our despair we clutched at this straw of chickenpox. It is well known that mumps encephalitis may anticipate the appearance of the parotid swelling; it may anticipate it by days or even a week or longer, and we wondered whether this man could possibly have had a varicella encephalitis with the appearance of the rash some time later.

Incidentally varicella encephalitis is a very fatal disorder.

Diagnosis.

Pancreatic islet cell tumours.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held on July 6, 1956, at the Anatomy Department, Medical School, University of Adelaide.

The Kinetics of Carbonic Anhydrase.

DR. W. W. FORREST discussed the kinetics of carbonic anhydrase. He said that the main purpose of the work was to study the activity of carbonic anhydrase at the high concentrations occurring in the red blood cells; there had been no previous studies in that concentration range. The reactions catalysed by the enzyme, the hydration of carbon dioxide and the dehydration of carbonic acid proceeded fairly rapidly in the absence of enzyme, and a rapid-reaction technique had to be used to observe the kinetics of systems of high enzyme activity. The activity of the enzyme was found to be proportional to its concentration up to concentrations giving several thousand times the uncatalysed rate. In that range, the data could be expressed in terms of the Michaelis theory; a value of K_m for carbon dioxide hydration of 0·020 M at 20° C. was obtained. At higher enzyme concentrations a limiting rate was reached, at which further addition of enzyme did not further increase the activity. The limit was a function of substrate concentration, and was not affected by the purity of the enzyme preparation. It was not predicted by the Michaelis theory, but there were not yet sufficient data to explain its cause. The enzyme concentration at which the limiting activity occurred was less than that found in the red cells, but the activity was greater than that necessary to account for the observed rate of elimination of carbon dioxide in respiration.

The Effect of Plant Root Exudates on Soil Microorganisms.

DR. A. D. ROVIRA discussed plant root exudates and their effect on soil microorganisms. He said that around plant roots there occurred a zone of soil, the rhizosphere, in which microorganisms were present in greater numbers than in soil beyond that zone. The mechanism involved in that stimulation of the microflora was studied by the use of peas and oats. The plants, which developed from surface sterilized seed, had been grown aseptically in quartz sand for ten and twenty-one days, after which the sand was washed free of material released by the roots. By centrifugation it was possible to separate the soluble fraction ("exudate") and the sloughed-off cell material and make approximate quantitative measurements. The results showed that at both ages peas released over double the amounts of soluble and non-soluble materials than did oats. In both plants the soluble fraction formed a higher proportion of the total material at ten days than at twenty-one days. Separation and identification of the aminoacid constituents by paper chromatography showed that peas released four to ten times the amount of amino material released by oats at corresponding ages. Peas exuded 22 amino compounds, of which the following were the most abundant: asparagine, serine, glutamine, homoserine, alanine (at twenty-one days) and threonine. From oats 14 amino compounds were detected, serine, lysine and glycine being the most abundant. Paper chromatography also revealed glucose and fructose in the exudate of both plants at ten days, but not at twenty-one days. Several substances which absorbed ultra-violet light and others which fluoresced under ultra-violet light were detected in the exudates, but were not identified. Studies on the effects of the exudates on bacteria isolated from soil showed that in several media the growth of the organism was increased by the incorporation of root exudate. That effect was greater for bacteria isolated from the rhizosphere than for bacteria found remote from the roots. The addition of root exudate to soils resulted in a rhizosphere-like effect, in that the Gram-negative bacteria were preferentially stimulated. Soils treated with root exudate were more active in the decomposition of readily decomposable materials than were untreated soils, but showed no greater activity in attacking the soil organic matter.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

INSUFFICIENT MEDICAL OFFICERS: GOVERNOR MACQUARIE TO EARL BATHURST.[1]

[From "Historical Records of Australia".]

28 April, 1814.

As on Occasions of Sickness in the Interior remote parts of this Colony, the Settlers and them Assigned to them from Government, are subjected to much Distress, even to the loss of their Lives, in consequences of having no Medical Aid near them, I beg leave to recommend that a few respectable Medical Men should be encouraged to come out to this Colony as Free Settlers, which I think it is probable many would be willing to do, were they assured of obtaining liberal Grants of land with the usual Indulgences without Government being put to further Expense, and their Establishment would be of great Importance in the distant Settlements.

I am sorry to be under the disagreeable Necessity of reporting to Your Lordship that Mr. Henry St. John Younge the last Medical Officer sent out to this Country and who arrived by the Earl Spencer Transport in October last is by no means a desirable Acquisition in the Line of his Profession. He is exceedingly ignorant as a Medical Man being almost destitute of Common Understanding and very low and vulgar in his Manners. From this Description Your Lordship will perceive that he is a very Unsuitable person to fill a situation of such Responsibility as that which has been assigned to him. I feel the more Necessity for making this Communication to Your Lordship in regard to Mr. Younge's Qualification, in the Hope that Your Lordship may direct a more minute Investigation of Medical Candidates for this colony to take place, and that they be certified as duly Qualified by the Army Medical Board previous to being appointed.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

THE MEDICAL BENEFITS FUND OF AUSTRALIA.

SIR: Some years ago this fund was established on the lines of an American scheme, because it was clear that if the medical profession did not suggest some concrete method of dealing with the rising costs of medical care, the Federal Government would bring into being a scheme of its own.

Indications of what that might mean could be gathered from experience of former proposals. So the Medical Benefits

Fund was established by the medical profession, individual doctors making contributions of ten pounds to cover initial expenses, publicity *et cetera*. Many contributed and many did not.

Those of us who did contribute this money did so in the firm belief that the proposed fund would be completely and permanently independent of government control. It is fair to assume that had sufficient money not been collected, the proposal may have died prematurely and similar schemes would have been instituted by the friendly societies or by insurance companies.

With the advent of the National Health legislation the fund has hit the jackpot, but in the process it has become decidedly a government agency. Whereas the potential patients, wanting cover for portion of their anticipated fees, subscribed voluntarily, now it is obligatory that they should be subscribers in order to participate in the government benefits.

This is something quite different, and there is surely a case that those original medical contributors wanting their money returned should receive a more sympathetic hearing than was given them on a previous occasion.

Further: (i) Doctors who did not make any initial contribution are benefiting equally with those who did. (ii) An original contributor who has given up practice for a full-time service position soon after the National Health scheme was established receives no benefit from his original contribution.

Admittedly the sum of ten pounds is very little in these days of devalued currency, but, speaking personally, I could purchase something useful for myself or my home which otherwise I would have to do without.

Yours, etc.,

S. SHINEBERG.

10 Bungaloe Avenue,
Balgowlah,
New South Wales.

August 14, 1956.

THE OLYMPIC GAMES, 1956.

SIR: For the first time in history the Olympic Games are being held in the southern hemisphere. It will therefore be realized how Australia has been honoured by the world of amateur sport. This particularly applies to Melbourne as the host city.

Each "Games" places a considerable burden on the medical profession of the country and city concerned. In the past, this burden has been treated as an honour, and the profession has risen to the challenge and gained distinction by the excellence of its achievement. Our efforts in war and peace have placed the medical profession of Australia in a position of distinction in the world. Here is another challenge for us to meet and fresh honour to win. Much time and effort are required by us—much of it is routine and unexciting, but it must be done. We confidently appeal to the profession in Melbourne in particular, and Australia in general, to provide the necessary honorary service to make the 1956 Olympic Games an outstanding success, and a model for the future.

The competitions commence on November 22 and end on December 8. Would any medical men or women living outside Melbourne, who will be available for some sessional work at the village or at sports venues, please notify the Chairman of the Medical Committee, c.o. British Medical Association (Victorian Branch), 426 Albert Street, East Melbourne?

A special circular is being sent to Victorian members of the British Medical Association, giving further details and requesting cooperation.

Yours, etc.,

GEORGE SWINBURNE,
President, British Medical Association (Victorian Branch).

H. CECIL COLVILLE,
President, Federal Council of the British Medical Association in Australia.

H. G. FURNELL,
Chairman, Medical Committee, XVIth Olympiad.

Melbourne,
August 3, 1956.

Public Health.

THE POISONS ACT, 1928 (VICTORIA).

THE following additions and amendments have been made to the *Poisons Act*, 1928, by proclamation in the *Victoria Government Gazette*, Number 735, of July 25, 1956.

Additions to Paragraph (1) of the Sixth Schedule to the Poisons Act 1928.

By His Excellency the Governor of the State of Victoria and its Dependencies in the Commonwealth of Australia, etc.

Whereas by sub-section (2) of section thirty-eight of the *Poisons Act* 1928 (No. 3748) as amended by section five of the *Poisons Act* 1930 (No. 3918) it is enacted that if it appears to the Governor in Council that any new derivative of morphine or cocaine or of any salts of morphine or cocaine or any other alkaloid of opium or any other substance or preparation of whatever kind is or is likely to be productive, if improperly used, of ill effects substantially of the same character or nature as or analogous to those produced by morphine or cocaine, the Governor in Council on the recommendation of the Pharmacy Board of Victoria may by Proclamation add the name of that derivative, alkaloid or other substance or preparation to paragraph (1) of the Sixth Schedule to this Act and declare that this Division shall apply to that new derivative or alkaloid or other substance or preparation in the same manner as it applies to the substances and preparations mentioned in paragraph (1) of the Sixth Schedule to this Act and the provisions of this Division shall apply accordingly:

And whereas the substances or preparations—

> Morphinan and derivatives of morphinan such as methorphinan, dextrorphan, laevorphan, levorphan, racemorphan, dromoran, 3-methoxy-N-methylmorphinan, dextromethorphan, laevomethorphan, levomethorphan, racemethorphan; and the salts, preparations, admixtures, extracts or other substances containing any proportion of morphinan or such derivatives—

were added to paragraph (1) of the Sixth Schedule to the *Poisons Act* 1928 by Proclamation of the Governor in Council dated the seventh of September, 1954, and published in the *Government Gazette* of the fifteenth of September, 1954:

And whereas the Pharmacy Board of Victoria has recommended that the name of the substance—

3-hydroxy-N-phenethylmorphinan—

be added to paragraph (1) of the Sixth Schedule to the *Poisons Act* 1928, as such substance is productive, if improperly used, of ill effects substantially of the same character or nature as, or analogous to, those produced by morphine or cocaine:

Now therefore, I, the Governor of the State of Victoria, by and with the advice of the Executive Council of the said State do by this my Proclamation add to paragraph (1) of the Sixth Schedule to the *Poisons Act* 1928 after the word "racemethorphan" in the said paragraph (1) the name of—

3-hydroxy-N-phenethylmorphinan—

And declare that Division 2 of Part III. of the *Poisons Act* 1928 shall apply to the substance—

3-hydroxy-N-phenethylmorphinan—

in the same manner as it applies to the substances and preparations included in the said paragraph (1).

Given under my Hand and the Seal of the State of Victoria aforesaid, at Melbourne, this seventeenth day of July, in the year of our Lord one thousand nine hundred and fifty-six, and in the fifth year of the reign of Her Majesty Queen Elizabeth II.

(L.S.) DALLAS BROOKS.

By His Excellency's Command,

E. P. CAMERON,
Minister of Health.

Amendment of Fourth Schedule to the Poisons Act 1928 (No. 3748).

By His Excellency the Governor of the State of Victoria and its Dependencies in the Commonwealth of Australia, etc.

By virtue of the powers conferred by section twenty-four of the *Poisons Act* 1928 (No. 3748) I, the Governor of the State of Victoria, by and with the advice of the Executive Council of the said State and on the recommendation of the Pharmacy Board of Victoria made on the 9th May, 1956, do by this my Proclamation amend the Fourth Schedule to the *Poisons Act* 1928 as follows, that is to say:

1. The following substances or preparations are hereby removed from Part II. of the said Fourth Schedule:

Diethylparanitrophenylthiophosphate or its isomers, whether known as such or as Parathion, Niram, E605, Phosphone, Paraphos, or by any other trade name, and all preparations and admixtures thereof.

Dimethylparanitrophenylthiophosphate or its isomers, whether known as such or by any trade name, and all preparations and admixtures thereof.

Hexaethyltetraphosphate, whether known as such or as H.E.T.P., Hexone, Demite, Hetraphos, or by any other trade name, and all preparations and admixtures thereof.

Tetraethylpyrophosphate, whether known as such or as T.E.P.P., Tephos, or by any other trade name, and all preparations and admixtures thereof.

2. The following substances and preparations are added to Part II. of the said Fourth Schedule:

Organic phosphate insecticides such as diethylparanitrophenylthiophosphate, Parathion, Niram, E605, Phosfone, Paraphos, dimethylparanitrophenylthiophosphate, hexaethyltetraphosphate, H.E.T.P., Hexone, Demite, Hetraphos, Tetraethylpyrophosphate, T.E.P.P., Tephos, Malathion, Diazinon, octamethylpyrophosphoramide, O.M.P.A., diethoxythiophosphoric ester of 2-ethylmercaptoethanol, Systox, E.P.N., or their isomers whether known as such or by any other name and all preparations and admixtures thereof.

Given under my Hand and the Seal of the State of Victoria aforesaid, at Melbourne, this seventeenth day of July, in the year of our Lord one thousand nine hundred and fifty-six, and in the fifth year of the reign of Her Majesty Queen Elizabeth II.

(L.S.) DALLAS BROOKS.

By His Excellency's Command,

E. P. CAMERON,
Minister of Health.

Additions to the Sixth Schedule to the Poisons Act 1928 (No. 3748).

By His Excellency the Governor of the State of Victoria and its Dependencies in the Commonwealth of Australia, etc.

By virtue of the powers conferred by section thirty-eight of the *Poisons Act* 1928 (No. 3748) as amended by the *Poisons Act* 1930 (No. 3918), I, the Governor of the State of Victoria, by and with the advice of the Executive Council of the said State and on the recommendation of the Pharmacy Board of Victoria made on the 13th June, 1956, do by this my Proclamation add to paragraph (1) of the Sixth Schedule to the *Poisons Act* 1928 the names of the following substances or preparations:

Dimethylthiambutene (3-dimethylamino-1, 1-di-(2-thienyl)-1-butene), its salts, and any preparation, admixture, extract, solution or other substance containing any proportion of dimethylthiambutene.

Ethylmethylthiambutene (3-ethylmethylamino-1, 1-di-(2-thienyl)-1-butene), its salts and any preparation, admixture, extract, solution or other substance containing any proportion of ethylmethylthiambutene.

Diethylthiambutene (3-diethylamino-1, 1-di-(2-thienyl)-1-butene), its salts, and any preparation, admixture, extract, solution or other substance containing any proportion of diethylthiambutene.

1,3 - dimethyl - 4 - phenyl - 4 - propionoxyhexamethyleneimine, its salts, and any preparation, admixture, extract, solution or other substance containing any proportion thereof.

4-morpholino-2, 2-diphenyl ethyl butyrate, its salts, and any preparation, admixture, extract, solution or other substance containing any preparation thereof.

And declare that Division 2 of Part III. of the *Poisons Act* 1928 shall apply to each of such substances or preparations so added in the same manner as it applies to the substances or preparations included in the said paragraph (1) of the Sixth Schedule.

Given under my Hand and the Seal of the State of Victoria aforesaid, at Melbourne, this seventeenth day of July, in the year of our Lord one thousand nine hundred and fifty-six, and in the fifth year of the reign of Her Majesty Queen Elizabeth II.

(L.S.) DALLAS BROOKS.

By His Excellency's Command,

E. P. CAMERON,
Minister of Health.

Amendment of Second Schedule to the Poisons Act 1928 (No. 3748).

By His Excellency the Governor of the State of Victoria and its Dependencies in the Commonwealth of Australia, etc.

By virtue of the powers conferred by section four of the *Poisons Act* 1928 (No. 3748), I, the Governor of the State of Victoria, by and with the advice of the Executive Council of the said State and on the recommendation of the Pharmacy Board of Victoria made on the 9th May, 1956, do by this my Proclamation amend the Second Schedule to the *Poisons Act* 1928 as follows, that is to say:

SECOND SCHEDULE.

LIST OF POISONS.

1st Part.

1. The article—

"Codeine and its salts, and all preparations and admixtures thereof containing 1·0 per centum or more of Codeine"

is hereby removed from such part of the said Schedule.

2. The following articles are hereby added to the 1st Part of the Second Schedule:

"Codeine and other morphine ethers such as benzylmorphine, ethylmorphine and pholcodine; and the salts, preparations, admixtures, extracts, solutions or other substances containing one part per centum or more of Codeine or such morphine ether.

Rauwolfia and its active principles and all preparations, admixtures and solutions of Rauwolfia or its active principles."

LIST OF POISONS.

2nd Part.

3. The article—

"Codeine. All preparations or admixtures of Codeine or its salts containing less than 1 per centum of Codeine"

is hereby removed from such part of the said Schedule.

4. The following articles are hereby added to the 2nd Part of the Second Schedule:

"Codeine and other morphine ethers such as benzylmorphine, ethylmorphine and pholcodine; and the salts, preparations, admixtures, extracts, solutions or other substances containing less than one part per centum of Codeine or such morphine ether."

Given under my Hand and the Seal of the State of Victoria aforesaid, at Melbourne, this seventeenth day of July, in the year of our Lord one thousand nine hundred and fifty-six, and in the fifth year of the reign of Her Majesty Queen Elizabeth II.

(L.S.) DALLAS BROOKS.

By His Excellency's Command,

E. P. CAMERON,
Minister of Health.

The World Medical Association.

THE PLIGHT OF DOCTORS IN HUNGARY.

We have received from the General Secretary of The World Medical Association the following statement on the plight of doctors in Hungary.

The attention of The World Medical Association has been directed to the degradation which has become the common lot of the professionally educated person in Hungary who,

prior to the current government regime, elected private practice rather than government employment. These doctors, engineers and architects who formerly employed free enterprise as a means of earning their living, are now reduced to a status of extreme poverty, and many of them are literally starving to death.

A specific instance, typical of many reported by first-hand observers, is cited here as an example.

A doctor, while earning his living through private practice, provided for his old age retirement by buying a modest home with apartments which he could rent, and invested his savings in insurance in United States dollars.

The Government nationalized all houses with apartments. Although legislation provides for compensation to the original owner, the Government refuses to pay anything to this doctor for his property. The insurance companies are also nationalized, and while insurance in United States dollars is continued and the State accepted all obligations associated with this insurance, it refuses to make any payments to the insured.

The doctor is eighty-three years old, deaf and almost blind. He is, therefore, unable to practise his profession. With all his retirement resources nationalized and the Government refusing to reimburse him for his house or pay him his retirement benefits, he and his seventy-four-year-old wife are assigned to live in a single small room built for a servant. Their only food each day consists of a lunch of soup and one vegetable, which is provided for them at the poor house.

Unfortunately, The World Medical Association is powerless to bring pressure to bear for the alleviation of this situation among the professional people in Hungary. However, by reporting this condition to the national medical associations, it is to be hoped that the member associations will be sufficiently moved by the inhuman and disgraceful status of colleagues in Hungary to urge their governments to bring pressure on the Hungarian Government through their United Nations delegations. Certainly situations of this type are not in accord with the International Covenant on Human Rights; nor do they promote the objective of The World Health Organization toward "the attainment by all peoples of the highest possible level of health".

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 48, of August 23, 1956.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointment.—Stephen John Lloyd (Acting Surgeon Lieutenant-Commander (for Short Service)) is appointed Surgeon Lieutenant-Commander, dated 1st May, 1956.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointment.—Peter Cameron Anderson is appointed Surgeon Lieutenant, dated 14th May, 1956.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

5/8054 Captain (provisionally) T. S. Samaha is appointed from the Citizen Military Forces, Royal Australian Army Medical Corps (Medical), Western Command, 22nd June, 1956, and to be Captain with a Short Service Commission for a period of one year.

To be Captains, with Short Service Commissions for a period of two years: 3/12026 Harry James Hodgson, 20th May, 1956, and 3/12027 Archibald Robertson, 1st June, 1956.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/39193 Honorary Captain A. J. J. Emmett is appointed from the

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 25, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	6(4)	5(5)	2(1)	..	..	..	..	..	13
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	2	..	..	..	..	..	..	..	2
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3(1)	5(5)	3(3)	..	..	..	..	..	11
Diphtheria	..	1(1)	2	..	2(2)	..	..	..	5
Dysentery (Bacillary)	..	2(1)	..	2(2)	1(1)	..	..	..	5
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	1	..	..	1
Infective Hepatitis	53(17)	24(12)	..	19(6)	3(2)	5(2)	4	2	110
Lead Poisoning	..	..	1	..	..	..	..	..	1
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	2	..	2
Meningococcal Infection	5(3)	7(7)	2(1)	..	..	3(1)	..	..	17
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	5(2)	2(2)	1	5(5)	..	1	..	..	14
Puerperal Fever	1	..	1	..	..	..	..	..	2
Rubella	..	15(10)	..	1(1)	5(5)	..	..	..	21
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	9(4)	..	2(2)	3(3)	3(3)	..	1	..	18
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	1(1)	..	..	..	2
Trachoma	..	..	..	..	3	..	..	..	3
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	23(16)	17(10)	9(2)	8(7)	15(13)	4(1)	1	..	77
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	1(1)	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Reserve of Officers, and to be Captain (provisionally), 29th May, 1956.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The notification respecting 3/101836 Captain (provisionally) G. R. McLeish, which appeared in Executive Minute No. 57 of 1956, promulgated in *Commonwealth Gazette*, No. 22, of 1956, is withdrawn.

3/101836 Honorary Captain G. R. McLeish is appointed from the Reserve of Officers, and to be Captain (provisionally), 15th February, 1956. To be Major, 21st June, 1956: 2/127019 Captain (Temporary Major) R. H. D. Bean. To be Captain (provisionally), 21st June, 1956: 3/50239 Gerard Reginald Warming.

Central Command.

Royal Australian Army Medical Corps (Medical).—To be Lieutenant-Colonels: Majors (Temporary Lieutenant-Colonels) 4/31907 R. A. Burston, 22nd June, 1956, and 4/31904 P. S. Eyles, 24th August, 1956.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/26544 Captain (provisionally) T. S. Samaha is appointed to the Australian Regular Army, Royal Australian Army Medical Corps (Medical), 22nd June, 1956. To be Captain (provisionally), 30th April, 1956: 5/26544 Tony Samuel Samaha.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

Eastern Command.—To be Honorary Captain, 21st June, 1956: John Sgouromallis.

Southern Command.—To be Honorary Captain, 8th May, 1956: Douglas Mackenzie Gee.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Dr. M. Finkelstein (Ph.D.), Lecturer in Endocrinology at the Hebrew University, Hadassah, Jerusalem, and a well-known authority on steroid estimating and steroids of the adrenal cortex, will lecture on "Steroids in the Adreno-Genital Syndrome" at the School of Public Health and Tropical Medicine, University of Sydney, on Thursday, September 20, 1956, at 4.30 p.m. This lecture has been arranged by the Post-Graduate Committee in conjunction with the Department of Veterinary Physiology in the University of Sydney.

Courses for Parts II D.G.O., D.L.O. and D.O.

The Post-Graduate Committee in Medicine in the University of Sydney announces that courses for Parts II of the diploma in gynæcology and obstetrics, the diploma in laryngology and oto-rhinology and the diploma in ophthalmology will begin in Sydney on November 19, 1956, for periods of three months each. Further details may be obtained on application to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-4498.

Royal Australasian College of Surgeons.

FACULTY OF ANÆSTHETISTS.

A MEETING of the Faculty of Anæsthetists of the Royal Australasian College of Surgeons will be held on Wednesday, October 10, 1956, at 8 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney. The subject will be "Vomiting in Relation to Surgery", and the programme will be as follows: "Preparation of the Vomiting Patient", Dr. E. A. Hedberg; "Management of Anæsthesia", Dr. G. M. Davidson; "Complications of Anæsthesia", Dr. L. T. Shea; "Post-Operative Vomiting", Dr. V. J. Kinsella. All medical practitioners are invited to be present.

Deaths.

THE following death has been announced:

LOWE.—Gordon Bradley Lowe, on August 31, at Croydon, New South Wales.

Notice.

THE Secretary of the Western Australian Branch of the British Medical Association wishes to draw attention to the fact that the Branch meeting announced for September 19, 1956, will not be held.

Diary for the Month.

SEPT. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 14.—Tasmanian Branch, B.M.A.: Branch Council.
SEPT. 14.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 19.—Victorian Branch, B.M.A.: Clinical Meeting.
SEPT. 20.—Victorian Branch, B.M.A.: Executive of Branch Council.
SEPT. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 25.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 *per annum* within America and foreign countries, payable in advance.